UNITED STATES OF AMERICA

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U.S. DEPARTMENT OF AGRICULTURE

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FOOD AND SAFETY INSPECTION SERVICE

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TECHNICAL MEETING

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THURSDAY
MARCH 24, 2005

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The meeting was held in the Federal Ballroom of the Holiday Inn on the Hill, 415 New Jersey Avenue, NW, Washington, D.C., Dr. David Goldman, Assistant Administrator, Office of Public Health Science, moderating.

PRESENT FROM USDA:
DAVID GOLDMAN, M.D., M.P.H., Moderator
DANIEL ENGELJOHN, Ph.D.
NEAL GOLDEN, Ph.D.
BARBARA J. MASTERS, D.V.M.
MERLE PIERSON, Ph.D.
CARL SCHROEDER, Ph.D.

ALSO PRESENT:
EDMUND CROUCH, Ph.D., Cambridge Environmental, Inc.
GREG PAOLI, B.A.Sc., M.A.Sc., Decisionanalysis Risk Consultants, Inc.

CONTENTS

P-R-O-C-E-E-D-I-N-G-S

9:06 a.m.

MODERATOR GOLDMAN: Good morning. I'm David Goldman. I'm the Assistant Administrator at FSIS for the Office of Public Health Science, and I want to welcome you here this morning.

We are here today to discuss two different risk assessments that were produced by our Risk Assessment Division, a risk assessment on *Clostridium perfringens* in ready-to-eat meat and poultry products and partially cooked meat and poultry products and another risk assessment on *Salmonella* in ready-to-eat meat and poultry products, both of which were developed to help guide FSIS development of stabilization and lethality performance standards as part of the ready-to-eat rule which was proposed in 2001.

I'll be your moderator this morning, and I'll get to some housekeeping notes at the end of my opening here. I want to say that the focus of this meeting is on the technical aspects of these risk assessments.

We'll present the risk assessments to you in great detail this morning, and we'll take this entire day as an opportunity for you both to hear what

we have to say about the risk assessments and, as importantly, to hear your questions and comments and, as best we're able, to provide answers to your questions during the course of the day.

The production of both of these risk assessments has been a high priority for FSIS. Our goal in producing in these risk assessments is to provide the best possible scientific basis for regulatory decision-making. So, again, today our focus is on the risk assessment and not on the agency's risk management thinking or policy proposals or plans.

We have just this day for the public meeting so I'd like to us focus if we can on the science and the data and how they have been used in the development of these two risk assessments.

In just a minute you'll get a welcome and a series of remarks that will help set the context for the discussions of these risk assessments. Let me now tell you that as a reminder, we are producing a transcript of this meeting so, if you will, take this opportunity to turn off your cell phones or put them on silent.

I've also learned that there's a possibility that some of the newer edition

Blackberries may interfere with the microphone equipment so if we have problems in the course of the day with some of the speakers, we may have to ask you to turn your Blackberries off.

Also, when you do come to the microphone at the appropriate times at the end of the morning and afternoon sessions, if you'll please use the microphones. The microphones will be turned on so that everybody can hear your questions and answers, but it will also assist us in producing a transcript of the meeting.

The bathrooms, if you have not found them already, are through the double doors at the back of the room, turn left and then turn right, and there are a set of women's and men's bathrooms there. I think that's the housekeeping items that I needed to mention at this point.

Now I'd like to introduce to you to provide the first welcome Dr. Merle Pierson, who was appointed as Deputy Under Secretary by Secretary Ann Veneman on February 4, 2002, and then recently on December 3, 2004, was appointed the Acting Under Secretary for Food Safety.

In this position, Dr. Pierson is responsible for overseeing the policies and programs

of the Food Safety and Inspection Service and he chairs the U.S. Codex Steering Committee, which provides guidance to the U.S. Delegations to the Codex Alimentarius Commission.

Dr. Pierson brings extensive scientific expertise to USDA. He is internationally recognized for his work on the hazard analysis and critical control points and research on reduction and control of food-borne pathogens.

He has co-authored or authored more than 100 journal articles, co-authored or co-edited seven books on food safety and presented numerous workshops on HACCP and food safety. Please join me in welcoming Dr. Pierson.

DR. PIERSON: Thank you, Dr. Goldman. Welcome and good morning to you. For those of you who weren't aware, Dr. Goldman recently came back from a 30-day deployment on the U.S. Navy ship Mercy off the coast of Indonesia to offer his medical expertise and professional support in the aftermath of the tsunami disaster.

One of Dr. Goldman's primary responsibilities was to assess some of the area's most devastated communities and make recommendations on how to improve the weakened infrastructure, especially in

the area of public health.

We're exceptionally proud of Dr. Goldman's service in Indonesia, and we're certainly glad to have him back with us in FSIS. We certainly appreciate his dedication to all facets of improving public health. So, I just think it's a tremendous service that he ?

We do have evidence of his presence there. He's got this flight uniform on and the goggles and the helmet and all that stuff so we should have had that up there. You could see him in his field dress so I guess that's one of the adventures you get for being in the public health service.

MODERATOR GOLDMAN: That's right, yes.

DR. PIERSON: But it is just a tremendous contribution. On behalf of USDA I want to welcome you to today's discussions on two of FSIS's recent risk assessments. I would also like to extend my appreciation to FSIS for hosting and organizing this meeting.

Today's forum is part of the continuing series of public scientific meetings that FSIS has held over the past three years. I believe we've had about a dozen of such meetings, and to us they're very, very important, and I'm sure that they're very important to those who are able to hear where we're

headed and ask questions about it.

Your input in these fora, yes, definitely, are very critical for the development of our food and safety policies. Advancing food safety is a tremendous challenge. It not only has accomplished the - its - it can only really be accomplished through the joint efforts with our stakeholders.

Regulation is an important component of our food safety system; however, it takes cooperation from all the stakeholders to make it a success, and we can have the policies but, for example, it's industry that quite frankly implements their control systems, validates their control systems so it just takes that full spectrum of individuals.

Equally important is implementing risk assessment and science-based policies to protect public health. Using this risk and science-based approach has been necessary to overcome many challenges the agency faced in the past several years. These are issues, for instance, relating to contamination by E. coli 0157:H7, *Listeria monocytogenes* and actually fairly recently as you all know the BSE issues. I mean we relied very heavily upon risk assessments and risk reduction in developing those policies.

To address these issues head on and to set our food safety system off on a course to improve public health significantly, we carefully developed a blueprint strategy from which we could guide all future policies and initiatives.

We had a five point strategy. I won't go through those. We've talked about those points. We've talked about those publicly many times. This original five point strategy provided us with a solid foundation and framework to assure that our food safety system would succeed and enhance public health protection.

At the same time it gave us latitude to refine our vision along the way. This is necessary since the crux of our public health challenge centers on combating biological, chemical and physical hazards that evolve and present new challenges, in addition to those that just seem to always be there persistently.

This is the reason why we published a visionary strategic plan two years ago, to complement our five-point strategy. The plan was entitled *Enhancing Public Health Strategies for the Future*, and it outlined a series of new and comprehensive science-based initiatives to better understand, predict and prevent microbiological contamination of those foods

under our regulatory authority.

And then last year we refined our visionary plan by publishing *Fulfilling the Vision Initiatives and Protecting Public Health* to evaluate the effectiveness of our strategy and to deal with outcomes associated with these initiatives.

I'm particularly proud of the work that FSIS has done over the past several years in using science to develop policies to improve safety and security of our meat, poultry, and egg product supply.

Through the use of comprehensive risk assessments and science-based policies, we're now finding smaller percentages of E. coli 0157:H7, *Listeria monocytogenes* and *Salmonella* positive regulatory compliance samples.

We've seen a break in the annual cycle of multimillion pound recalls. Please, let's not have large, huge recalls. Hopefully we've broken that cycle. We've seen that that cycle has been broken, and we want to continue addressing these areas very aggressively.

These examples represent major advancements in areas which provided troubling challenges in the past; however, our work is not finished.

We need to follow through on recent progress and continue to drive down food borne illness rates even further by continuing the application of risk and science based principles in all of our initiatives. This is why I'm very interested in the discourse and outcome of today's meeting.

The risk assessments on *Clostridium perfringens* in ready-to-eat and partially cooked meat and poultry products and the impact of lethality standards on salmonellosis from ready-to-eat meat and poultry products are the latest tools the agency is pulling from our tool chest to base any future decision-making on the best available science.

The presentations you'll hear and the input you provide will go a long way in improving the safety of some of our nation's most highly consumed and popular food products.

Certainly there will be many questions about both of the risk assessments, the results, and the next steps by FSIS. Members of our leadership team as well as a couple of presenters from outside the agency are here today and each will be explaining the different aspects in more detail.

Once again I want to thank you for your participation at this meeting, and I will look forward

to the discussions that you have today and your input. Thank you very much.

MODERATOR GOLDMAN: Let me now introduce to you Dr. Barbara Masters who is the Acting Administrator of the Food Safety and Inspection Service and, as such, is responsible for managing the day-to-day activities of USDA's food safety and food security activities.

She was previously the Deputy Assistant Administrator for the Office of Field Operations. She's been with FSIS for about 15 years serving in a variety of positions in the field and at headquarters.

At the Technical Service Center, she served as Director of Slaughter Operations Staff and as the Branch Chief for Processing Operations. She was a Staff Officer for the Slaughter Operation Staff and the Technology Transfer and Coordination Staff.

She has also served as an Inspector in charge in the livestock slaughter and processing establishment. Dr. Masters has a Doctorate of Veterinary Medicine from Mississippi State University and served a food animal internship at Kansas State University. Please welcome Dr. Masters.

DR. MASTERS: Thank you, Dr. Goldman, and good morning to all of you. We certainly welcome you

from the Food Safety and Inspection Service and appreciate your attendance at this very important meeting.

We at FSIS, as Dr. Pierson indicated from the Office of Food and Safety, certainly recognize the importance of having these type of public forum to discuss things such as these risk assessments and to gain public input on risk assessment and policy development by the agency.

Certainly we at the agency are interested in hearing from you on our risk assessment that we're going to be discussing today. Risk analysis, including the risk assessment, risk management, and risk communication component, is one of our agency top priorities right now. We believe that risk assessments provide critical information that allow our risk managers to identify steps that can lead to public health improvements.

We recognize that risk assessments can lead to regulatory changes but we also recognize that they can lead to other opportunities such as educational initiatives or even to allow us to identify data gaps that can allow us to look for recommendations in the area of research needs.

So we use risk assessments in many ways as

an agency. In fact we have all ready completed many risk assessments. We've completed risk assessments for *Salmonella enteritidis* for eggs, E. coli 0157 for ground beef, *Listeria monocytogenes* for ready-to-eat meat and poultry products, and we've contracted with the Harvard University's School of Public Health for a risk assessment on BSE.

We've used the results of these risk assessments to develop food safety risk management strategies to further protect public health. We also recognize that we can't just do these risk assessments, that we must continually update our risk assessments as we get newer scientific information.

In fact, we've been able to include updates such as including a production volume in our 2003 *Listeria* assessment through a survey that we cleared through the Office of Management and Budget. We've also updated our SE risk assessment on eggs by including more baseline information, and we're also working with the Harvard University to update our risk assessment on BSE.

We use these updated risk assessment to provide our scientific basis for future decision-making. So we do recognize the value of risk assessments, gaining newer scientific information,

oftentimes by working with you all so that we can continue to move forward as an agency and our policy making.

We do believe that through these type of productive forum that we can continue to gain information. We are dedicated, as we know you are, to improving public health.

During this meeting you'll hear from our FSIS representatives as well as from outside representatives, and, as Dr. Goldman said, because we have so much information to talk about, we're going to focus at this meeting on the risk assessments themselves.

You will have opportunities through May 9 to provide feedback to the agency. We recognize that the risk assessments are not just a quick, easy read. That's why we feel like it's important to have these public meetings so that we can try to walk through the documents, provide executive summaries to you, and provide an opportunity so that you can ask our agency those kind of questions that are useful to you so that you can give us the best input.

We do value the input and comments that you give to us, and we recognize if we just left you with the risk assessments, we may not get the best

comments back.

Please use this forum today to focus on the information we're giving you so that through May 9 you can get the best comments back to us. That is what we're seeking from you, and we do value that time that you take to use today to best think about very critically the kind of information that is useful to give back to us as an agency so that we can move forward.

We appreciate the time that you're taking today as well as the time you take between now and May 9 to give us those very thoughtful comments. Thank you.

MODERATOR GOLDMAN: All right, if you will pull out your agenda, I want to walk you through that very briefly. What you'll notice is the morning and afternoon sessions are set up the same way, and that's to reflect the fact that there are two separate risk assessments, as I mentioned before.

The way we've devised this agenda you will hear from Dr. Engeljohn, whom I'll introduce in just a minute, the regulatory policy context for these risk assessments, then you'll hear from one of the risk assessment staff members about the public health context, and then you'll hear from, in this case, two

contractors to the Risk Assessment Division who have actually done the risk assessments for the agency.

You'll see there is ample time after each of the morning and afternoon sessions for you to ask your questions, and I will remind again to please use the microphones for the questions and comments. We have an hour-and-a-half lunch from 11:30 to 1:00 and we'll start promptly after lunch at 1:00 with the second risk assessment.

Let me introduce to you Dr. Daniel Engeljohn who is the Deputy Assistant Administrator for the Office of Policy, Program and Employee Development at FSIS. He oversees the risk management activities associated with meat, poultry, and processed egg products and manages the staffs that develop regulations and policies that are associated with inspection procedures, data analysis, and performance standard strategies.

Dr. Engeljohn has worked at USDA for 24 years. He also serves as an adjunct Assistant Professor of Nutrition on the Graduate Faculty at Howard University and teaches undergraduate and graduate courses on human nutrition.

He holds a B.S. and M.S. degree in animal science from the University of Illinois and has a

Ph.D. in nutrition from Howard University here in Washington, D.C. Please welcome Dr. Engeljohn.

DR. ENGELJOHN: Good morning. I'm going to walk you through some of the policy and risk management questions for consideration with regards to these risk assessments today.

Here's a bit of an overview of what I'll present in this first presentation. I'll talk risk management and risk managers, the background on the proposed rule, risk management questions regarding *Clostridium perfringens*,and then give you a summary.

I thought it was important to start out with a definition. I represent the policy side of the agency as opposed to the public health science side of the agency, and so from my perspective I represent risk management.

Risk management as defined in a Codex document is the process, distinct from risk assessment, of weighing policy alternatives in consultation with interested parties, and selecting appropriate prevention and control options.

There are eight general principles including protecting human health is the primary consideration. Risk management should follow a structured process. Risk management should ensure

effective consultation with relevant interested parties, which is a primary reason why we're here today, and risk management should ensure effective interaction with risk assessors.

As a risk manager, this would be defined as a national or international governmental organization with responsibility for microbiological risk management. This also was taken from a Codex documents.

Risk managers then can set the food safety objectives. Risk managers and industry may in fact set the performance objectives, performance criteria, as well as the microbiological criteria.

With regards to background on stabilization policy, stabilization for those of you who are not familiar with that term, we use that term for describing cooling of cooked products.

We issued a final rule on cooked meat patties, roast beef, and cooked poultry in January of 1999. In that regulation, we required that those products listed, exposed to heat could have no more than one-log growth of *Clostridium perfringens* during stabilization, and there could be no multiplication of *Clostridium botulinum*.

Shortly thereafter the agency worked

another proposed rule to bring in all of the other ready-to-eat products that were not presently regulated in the January 1999 regulation. That rule encompassed all of the other ready-to-eat products. Within that proposed rule, we added the same stabilization requirements as we had required for the cooked meat patties, roast beef, and cooked poultry from before.

We did receive a number of comments on this particular rulemaking, and, in fact, the comments indicated that the stabilization performance standard was too restrictive. With that information, the agency then decided that we needed to relook at what we had done with the proposed rule.

The design of the stabilization performance standard was in fact based on longstanding policy that the agency had in place for many years prior to ruling in January of 1999.

It was also a practice that the agency believed most industry members would be able to meet. In any case, we did put together that proposed rule based on existing practices. Based on the comments then we asked our risk assessment group within the agency to develop a risk assessment so that we can look at the process of how to more completely and

fully address this standard.

The risk assessment is a scientifically based process consisting of hazard identification, hazard characterization, exposure assessment and risk characterization. So therefore the risk managers in the agencies formulated the following questions in order to pose to the risk assessors in the design of the risk assessment.

I see my slides don't actually fit on this page here. The first question was what is the impact on the probability of human illness if the allowable growth of *Clostridium perfringens* is raised from one-log during stabilization to two logs.

The second was what is the impact on the probability of human illness if the allowable growth of *Clostridium perfringens* is raised from one-log during stabilization to three logs. The third question is what would be the relative growth of *Clostridium botulinum* for each of these stabilization standards.

In summary, FSIS is the public health regulatory agency responsible for ensuring the safety of the meat, poultry, and egg products. We recognize the importance of science and informing policy.

We seek to have a transparent process.

That's part of why we're here today, as well as we seek to involve interested parties, to get your input on the risk assessment that we're presenting. With those risk management questions, we'll now move to the next portion.

MODERATOR GOLDMAN: Thank you. Now we'll move into a more specific focus on *Clostridium perfringens*, and Dr. Neal Golden will present an introduction, an overview of the risk assessment and again the public health context for this risk assessment.

Dr. Neal Golden has served as a risk analyst in the Food Safety and Inspection Service Office of Public Health Science for the past three years. He graduated from Tufts University Sackler Graduate School in Boston, Massachusetts, with a Ph.D. in immunology.

He is currently involved in several risk assessment projects in the agency, including *Salmonella* species in raw beef and poultry and E. Coli 0157:H7 in ground beef. Dr. Golden?

DR. GOLDEN: Great, I appreciate the introduction. So thank you all for coming. The bulk of my presentation will be on providing a brief overview to the microbiology of *C. perfringens* and the

epidemiology of *C. perfringens* food borne illness, the risk assessment model, the answers to the specific risk management questions as well as the peer review comments will be presented by Dr. Edmund Crouch following the break.

Now by way of overview, I am going to give a brief portion of the background of the risk assessment, the microbiology of *Clostridium perfringens*, the epidemiology of *Clostridium*'s food borne illness. By that way I hope to give a context and more of the background to this current risk assessment, and then finally I will summarize the slides.

Okay. So by way of background, so during processing of ready-to-eat and partially cooked foods raw meat and poultry that are destined to become such commodities as ready-to-eat and partially cooked are heat-treated so a lethality step is applied and then cooled in a process known as stabilization.

Now spores from pathogenic organisms such as *Clostridium perfringens* and *Clostridium botulinum*, excuse me, may be activated by the heat treatment and germinate into vegetative cells that are capable of growing in such commodities.

Now the current USDA stabilization

performance standards states that no more than a massive 1-$\log_{10}$ growth of *C. perfringens* and so that's a factor of ten and no growth of *C. botulinum* is allowed during the production of processed meat and poultry products.

In response to public comment on the proposed rule, FSIS initiated the planning and the development of this risk assessment. I'll now give you a brief background on the microbiology of *C. perfringens*. Excuse me.

C. perfringens is a Gram-positive spore-forming bacteria that grows well in meat under anaerobic conditions. So these are conditions in which no oxygen is present. Excuse me.

C. perfringens is ubiquitous within the environment. It's present in high levels within the soil. It's present in dust. Excuse me. It's present in the gastrointestinal tract of animals and in humans as well.

C. perfringens grows optimally within the range of 43 degrees Celsius and 47 degrees Celsius. In this range, it can grow quite rapidly, and in addition it has a broad growth range in between 12 degrees C and about 52 degrees C.

Now there are many different types of *C.*

perfringens; however, the risk assessment only focuses on those *C. perfringens* that can cause *C. perfringens* food borne illness.

Those are *C. perfringens* that are type A and are capable of producing the enterotoxin known as *C. perfringens* enterotoxin or CPE; therefore, this risk assessment only focuses on *C. perfringens* that are type A and *C. perfringens* that are enterotoxin positive.

Now in this slide I am going to review the pathogenesis of *C. perfringens,* and I first want to orient you on this slide. Now above the hatch mark is what happens to *C. perfringens* in the food, and below the hash mark is what happens to *C. perfringens* once it's consumed and enters the gastrointestinal tract of a human.

Now meat and poultry products can be contaminated with *C. perfringens* spores and C. *perfringens* vegetative cells. So during the processing, a way to eat meat and poultry products that are destined to become RTE are partially cooked when a heat lethality step is applied.

Vegetative cells are killed; however, spores are activated to germinate into vegetative cells and could grow to high levels if stabilization

is inadequate or if time and/or temperature abused at later steps throughout the foods processed continual.

Now it's important to note that unlike pathogens such as *Staphylococcus aureus*, *C. perfringens* does not produce a preformed toxin within the food and rather it produces a toxin in the gastrointestinal tract of a human. So it's really an infection and not an intoxication.

So when a product is consumed that contains *C. perfringens*, that is vegetative cells, it makes its way through the gastrointestinal tract to the small and large intestine. Now it's actually thought that this harsh environment results in a morphological and a physiological change of *C. perfringens* vegetative cells into spores.

So, of course, at this moment so now I'm talking about what happens underneath the hashed life. Now the vegetative cells go from capable of growing to a spore state where they are not capable of growing and during the process of sporulation the toxin is produced in the gastrointestinal tract.

Therefore, the presence of large numbers of *C. perfringens* vegetative cells in the food may result in large or a high level of *C. perfringens* enterotoxin, food toxin within the gastrointestinal

tract that could lead to illness.

As a result the risk assessment primarily focuses on how vegetative cells in spore populations change from the processing plant to the consumer. Now that we've reviewed the microbiology of *C. perfringens*, I'd now like to speak about the disease that this pathogen results in.

Mead and colleagues at the CDC estimated that approximately a quarter of a million illnesses 41 hospitalizations and seven deaths annually are associated with *C. perfringens*. Additionally, Mead and colleagues also estimated that *Clostridium botulinum* causes 58 illnesses, 36 hospitalizations and four deaths annually.

Now *C. perfringens* and *C. botulinum* share a common food vehicle, and that is meat and poultry. Additionally, during stabilization of RTE and partially cooked foods, *C. perfringens* and *C. botulinum* could grow in numbers and become potentially significant to public health.

In terms of the disease characteristics, the symptoms that are typically associated with *C. perfringens* are diarrhea, nausea, and abdominal pain. The incubation period ranges from approximately eight to 24 hours, and this is actually relatively quick

compared to other bacterial food borne illnesses; however, *C. perfringens* is very much a mild illness and is self-limiting, lasting at most two to three days.

However, severity can vary, yet it's important to keep in mind that severe illness and chronic sequelae are rarely, if ever at all, associated with *Clostridium perfringens* food borne illness.

Now what can the *C. perfringens* CDC outbreak data to tell us about the epidemiology of this pathogen. Well as I mentioned before, the most common implicated food vehicle for *C. perfringens* food borne illness is meat and poultry.

Over a ten-year period, from 1999 - excuse me, from 1990 to 1999, 153 *C. perfringens* outbreaks were recorded. The majority were associated with meat and poultry prepared from the raw products.

In other words, epidemiology is not associated with RTE or partially cooked foods. In fact only one outfit has even confirmed as having been caused by a RTE product and this is turkey loaf.

Additionally, the outbreak data tells us that in those outbreaks where a contributing factor to the outbreak was recorded, the majority of *C.*

perfringens outbreaks is associated with improper holding. This occurs at institutional places such as hospitals, nursing homes, prisons, schools, et cetera.

The criteria for *C. perfringens* outbreaks includes presence of ten to the fifth *C. perfringens* organisms per gram in implicated food. Once again this suggests that large numbers of *C. perfringens* in vegetative cells are needed to cause illness or presence of 10^5 *C. perfringens* organisims per gram in stool from two or more ill patients or the demonstration of the enterotoxin in the stool from two or more ill patients.

Lastly, approximately 250 outbreaks involving approximately 15,000 cases were reported to the CDC over a 14-year period. Now I'd like to summarize my presentation. *C. perfringens* is estimated to be the fourth most common cause of food borne illness in the United States as estimated by Mead and colleagues.

C. perfringens and *C. botulinum* food borne illness are associated with meat and poultry and could become a hazard in RTE and partially cooked products if stabilization is inadequate.

Now to control the growth of *C. perfringens*, FSIS regulates the critical control

allowable limit of *C. perfringens* at the processing plant.

Lastly, in response to public comment, the agency has developed and completed a risk assessment to evaluate the public health impact of the current USDA stabilization performance standard. Great, so thank you very much.

DR. GOLDMAN: We're exactly on time and have a 15-minute break. We'll resume at ten o'clock with the presentation of the risk assessment.

(Whereupon, the above-entitled matter went off the record at 9:46 a.m. and resumed at 10:04 a.m.)

DR. GOLDMAN: I think we'll get started with the next portion of the agenda. At this point we will hear a presentation on the risk assessment that was done for *Clostridium perfringens*.

Let me introduce for you the scientist who conducted the risk assessment, Dr. Edmund Crouch, who has published widely in the areas of environmental quality, risk assessment and presentation and analysis of uncertainties. He has co-authored a major text in risk assessment, Risk Benefit Analysis.

Dr. Crouch also serves as an expert advisor to various local and national agencies concerned with public health and the environment and

has served on two National Academy of Science Committees.

Dr. Crouch holds a B.A. in natural sciences and a Ph.D. in high-energy physics both from Cambridge University and the United Kingdom. He is currently a senior scientist at Cambridge Environmental, Inc. in Cambridge, Massachusetts. Please welcome Dr. Crouch.

DR. CROUCH: Thank you. Good morning, ladies and gentlemen. Well, for the next 45 minutes or so ? oh, now I see it's only 40 minutes, I'm going to speaking on the *Clostridium perfringens* risk assessment in ready-to-eat and partially cooked meat and poultry products.

I'm going to quickly here give an overview of what I'm talking about and start by introducing the context from my point of view, tell you the risk management questions that the risk assessment was designed to answer, then quickly go through the risk assessment itself, talking about the foods that are modeled, the conceptual model used in the risk assessment and how the foods were transported through that model, the dose response assessment that we did for this and then go on to summarize the results in the form of the answers to the risk management

questions, discuss a couple of the risk factors that turn up in the risk assessment.

I'm also going to give one example of the what-if scenarios that were done in the risk assessment, and I'll tell you why we had to do those.

I'll quickly summarize peer review comments and then summarize this whole talk. The context that we started with in the risk assessment is that during processing of ready-to-eat and partially cooked meat and poultry products - thank you. All right, now I'm not tied down quite so much. Thank you.

During processing of ready-to-eat and partially cooked meat and poultry products, raw meat and poultry are heat-treated in what's called the lethality step and then cooled in the stabilization step.

Spores from pathogenic organisms and in particular here *Clostridium perfringens* and *Clostridium botulinum* may be activated by the heat treatment and germinate into vegetative cells and those cells may grow at temperatures that are permitted for that growth during the stabilization in particular but then subsequently during the transport of food from the processing plant, storage and

transport from the processing plant and storage at home and during cooking even.

C. perfringens and *C. botulinum* spores may be present in the raw meat and poultry that are used to produce the ready-to-eat and partially cooked foods. The risk assessment I'm looking at here investigates *C. perfringens* with respect to stabilization performance.

That is, what is the effect of stabilization performance on human health when we track *C. perfringens* from the raw meat all the way through to people eating the foods. The USDA standard from 1999 requires that there's no more than 1-$\log_{10}$ growth ? that's a factor 10 growth for some ready-to-eat and partially cooked food products.

I assume that everybody here is familiar with what the $\log_{10}$ notation means. 1-$\log_{10}$ is a factor of ten; 2-$\log_{10}$ is a factor of 100; 3- $\log_{10}$ is a factor of 1000, and so on. The USDA standard also requires no growth of *C. botulinum*, and in the risk assessment we examine what effect changes in the CP standard would have on *C. botulinum.* That was one of the risk management questions essentially.

The explicit risk management questions were what's the impact on the probability of human

illness if the allowable growth of CP is raised from 1-$\log_{10}$, a factor of ten, during stabilization, and what would be the relative growth of *C. botulinum* in those conditions relative to the growth of CP for each of those stabilization standards, if they were assessed as ? if they were imposed as stabilization standards.

Now the risk assessment I'm going to quickly discuss the foods that were modeled, how the exposure assessment was done, and the dose response assessment that was applied, and then tell you the results that come out of it. We started by looking at what foods are eaten, and to do this we used the survey, the Continuing Survey of Food Intake by Individuals, CSFII. And from these, from the CSFII, we examined the foods that were looked at in the ? or observed that people were eating in CSFII and selected out the 1625 types of food in that survey that contain meat or poultry.

The CSFII contains descriptions and estimates of quantities for each of the foods and beverages that participants ate or drank, and this is during the period 1994 to '96 and 1998 that this survey, the data that we used, was taken. Each food in the survey has a recipe, and that recipe was what

we looked at to look at the meat and poultry content of it from that recipe as well and used it in the risk assessment. There's sometimes some information on cooking and preparation methods, as well, but we weren't able to use that; it's insufficient. These meat and poultry product foods were obtained by searching all the foods on various keywords to pick out meat and poultry.

From these entries, various foods were removed because they wouldn't support the growth of *C. perfringens* so that foods that are determined to be shelf-stable were removed, and foods with a high salt concentration that prevents the growth of *C. perfringens* were also ? that turned out removing nothing because people don't generally eat things with high salt content, not meat and poultry products, anyway. Foods which contain both nitrites and more than 3% salt were excluded because, again, they don't support the growth of *C. perfringens* either.

Raw commodities were excluded because we're looking at ready-to-eat and partially cooked foods, not raw commodities. So we were left with 607 types of food that could correspond to ready-to-eat and partially cooked meat and poultry products, and from these 607, there were actually 26,548 servings

within CSFII, and we used all of those in the risk assessment. Those foods were considered representative of partially cooked ? of ready-to-eat and partially cooked foods, meat and poultry products for use in the risk assessment. It was - they were considered representative of what Americans eat.

Now the layout of the risk assessment itself is an attempt to a-plant-the-fork risk assessment in which we modeled the growth, survival, and death of vegetative cells and spores from just after the heat step from - sorry, from after the heat step at the processing plant to consumption by the consumer.

What we're doing is we're tracking individual surveys through this process - individual servings of food through this process. Here we have a complicated chart, which probably some of you in the back can't read. It's in the same diagram as in the risk assessment itself.

We start with by breaking up the process into three modules, essentially. We start with processing. Raw materials come into the processing plant and are subjected to a heat step in ready-to-eat foods.

This kills the vegetative cells that are

present in the food at this point; however, it activates some spores, some of the - the majority of the spores would get activated in these heat step and they would germinate to produce new vegetative cells.

For partially cooked foods, the vegetative cells and spores are considered in the risk assessment to be unaffected by any partial cooking that is done in that step.

The reason we made that assumption is that we could find no information whatsoever on what effect such a heat step - such a partial cooking has on vegetative cells and spores.

Subsequently, stabilization is performed to reduce the temperature after the cooking and reduce the temperature below temperatures at which *C. perfringens* can grow.

During this procedure, vegetative cells can grow. Spores, any remaining spores that weren't activated from ready-to-eat or were present in the partially cooked foods are unaffected by this step and remain there.

What we did here is we assumed that during this step there would be a defined growth of CPE, of *C. perfringens*, of well one, two, or three $\log_{10}$ included what we did it for several other growth rates

as well.

We couldn't model what actually happens in processing plants. We just do not have the information for that so we examined what would happen if there was a certain amount of growth.

From there, foods - food servings are assumed to proceed through storage and then preparation. In that procedure - in that process, some of the spores might germinate to vegetative cells. There's an indication that this procedure can occur even under extremes of temperature; even in freezing conditions some spores can germinate.

In the model we put a small fraction of spores germinating at the beginning of this process to account for that process.

During storage at plant and subsequently at retail and at home, depending on the temperature, primarily on the temperature of storage, vegetative cells or *C. perfringens* will either grow or die or be pretty well unaffected. Spores will be pretty well unaffected.

We tracked what happens to the vegetative cells as they grow or die during storage at plant or retail or storage at home. Finally at - in the home or final use, we have preparation of the food serving,

and we here looked at three possibilities. First is reheating of the food.

This is the most of the ready-to-eat and partially cooked food would be reheated. During this reheating vegetative cells would initially start to grow as you start to warm the food up and subsequently die as you took it a high enough temperature above about 50 Centigrade.

By this point, we don't have to track spores anymore because we're only interested in the vegetative cells because we're interested down here in how many vegetative cells do people eat in each serving that they eat of ready-to-eat and partially cooked foods.

So we reheat and track what happens as the vegetative cells grow and die. Alternatively, some fraction of ready-to-eat foods are eaten cold so that any vegetative cells and spores at this point are eaten immediately. Some small fraction of servings are hot held. In this case they are heated up in what we called another cooked step. It's just a reheating step.

This reheating will kill vegetative - any vegetative cells are - that are present at this point; however, any remaining spores will get activated and

then during subsequent hot holding these spores which have been activated and germinate to vegetative cells will themselves either grow further or die off, depending on the temperature of hot holding.

We don't, again, track spores by the time we get to the hot-holding because we're only interested here in vegetative cells so that what people eat are the vegetative cells.

Now throughout this we've evaluated what happens based on various bits of data that come from literature or from industry surveys or from regulatory surveys. Here we got temperature from an FDA survey for example. All these data are incorporated in the risk assessment.

Now the modeling in the risk assessment is tracking individual surveys - individual servings through this process. The modeling of surveys - of servings, excuse me, takes account of the numbers of spores and the numbers of vegetative cells initially in the serving.

It takes account of both the variability of that from serving to serving and how uncertain we are about it and then so all the subsequent steps from this information we've evaluated what happens taking account both of how it varies from serving to serving

and how uncertain we are of what is happening in that step so that we're tracking variability and uncertainty and what happens to these spores throughout this whole modeling exercise.

The way we did that is by Monte Carlo techniques so that we can estimate the variability and uncertainty in the results that we get down here and how much people eat so we can estimate from serving to serving we get variation. It looks like the battery has run out.

We get variation from serving to serving and how many cells there are - how many vegetative cells are eaten. We make - we also get an estimate of how uncertain we are in the numbers of vegetative cells that are eaten.

The servings that go through this process in the Monte Carlo assessment are randomly selected from those 26,548 that we got out of the CSFII so we take account of the full range of amounts of meat and amounts of salt for example in each serving and track those correctly.

These are ? the variabilities and uncertainties are represented by probability distributions, which are in turn obtained from the literature and analysis of the literature that we

found and the information that we found.

A large part of these 304 pages of the risk assessment is dealing with all of these analyses to find out what are the uncertainty and variability distributions to use in these processes. Once we get it into humans, once humans eat it, we've got to figure out what happens. Are they going to get ill?

For this we've evaluated dose response curve based on four human clinical trials that were performed or reported in the literature somewhere between 1954 and 1971. This risk assessment is based - is evaluating illness in humans so we concentrated on CP type A, enterotoxin-positive and evaluated just those.

In order to do this we've got to have dose response curves, and it was - the clinical trial data was evaluated, and it was found that there's a huge random - apparently random effect between strains - well not random, individual strains of CP vary substantially in their propensity to cause diarrheal illness.

So we evaluated the dose response curves using a pretty simple dose response curve for each individual strain but put in what's called a lognormal random effect model for the between strain effect

taking account of the variation in the potency of these - of each strain to cause diarrhea.

Out of this, for example, from the model, from fitting these dose response curves, consumption of about 5 x 10^7 CP vegetative cells results in a one percent attack rate on average.

What means really is that the probability of anybody getting diarrhea if they ate 5 x 10^7 vegetative cells of a random strain of *C. perfringens* type A enterotoxin-positive is about one percent.

So we have how many cells, how many CP vegetative cells - *C. perfringens* vegetative cells get into people? We have an estimate from the dose response curves of what the probability of causing diarrhea is from that, and again we've got - it's a probabilistic approach.

We've got uncertainty and variability. Uncertainty in this case, how uncertain we are about the dose response curves; variability is between strains in this case.

From that we can estimate what's the probability of for each serving that we model going into people that they eat. We can estimate the probably of it causing diarrheal illness and so use this model to answer the risk management questions.

We can evaluate in the model what are the important risk factors, and we can also evaluate what happens if we change assumptions in our risk management, in our risk modeling, because some of the modeling we have to make assumptions because there are just too few data to get good estimates for them.

So the first risk management question which is what is the impact on the probability of human illness if the allowable growth of CP is raised from 1-$\log_{10}$ during stabilization to 2- or 3-$\log_{10}$. Remember in the risk assessment, we're not quite matching that.

We're not looking at allowable growth. We're looking at actual growth. We are going to also question what happens if the actual growth is 1-$\log_{10}$ or 2-$\log_{10}$ or 3-$\log_{10}$ and so forth.

Now this is what the modeling estimates. The change in growth, and this is the $\log_{10}$ growth along the bottom axis, during stabilization from one to two to three results in a median increase by a factor of 1.21 and 1.57 respectively for two and 3-$\log_{10,}$ but here's the curve for other growth, increase in annual diarrheal illness.

You can see there's a smooth increase as you increase the growth and the plot up here is

illnesses per - it says million servings - that's illnesses per million servings - of diarrheal illnesses.

Our estimate for the baseline is two illnesses per million servings, 1-$\log_{10}$ and that would increase by a factor of 1.21 to 1.57 - 1.21 at 2-$\log_{10}$ growth and 1.57 at 3-$\log_{10}$ growth, and we've got a smooth curve up here.

The vertical bars on this represent the uncertainty that we estimate from our modeling, and that's a 90 percent - where if all the assumptions of the model are correct then we're 90 percent sure that the true value would lie within the - within that - the range of those error bars, and the whole curve will move up and down those error bars as the uncertainty varies.

That's just showing how large the uncertainty is. It's about a factor of two uncertainty if all our assumptions are correct. We are at Monte Carlo modeling. There's a numerical uncertainty as well, and that's given by the small error bars there to - just to show that we've sampled enough time - did enough runs in our Monte Carlo.

We also looked at the total number of annual *C. perfringens* illnesses estimated from this.

Looking at the epidemiology of the 153 CP outbreaks in 1990 to 1999 as Dr. Golden said, only one has been confirmed from RTE product, and that was turkey loaf.

The majority of outbreaks occur in institutional settings and are thought to be the result of meat prepared from raw rather than RTE product.

Mead et al. in 1999 based on observations in *Salmonella* illnesses so extrapolating from *Salmonella* to account for underreporting estimated quarter of a million annual CP illnesses for all food sources.

What the model is estimating for RTE and PCF, 1-$\log_{10}$ growth is a best estimate of about 113,000 illnesses per year in the US, and we're uncertain on that, at least by a factor of two.

The factor of two comes from the uncertainties that we know about. There are also uncertainties that we don't know about, and I've listed them in the risk assessment, and there's quite a long list, which will increase that uncertainty.

So we're treating this model as a tool to evaluate the effective interventions rather than to predict the absolute number of illnesses. We cannot confirm that that is the absolute number of illnesses,

for example, because of insufficient information.

Some of the contributing factors - the risk factors that we analyze by using the risk assessment model are the risk management question itself, that is what's the effect of stabilization at food processing plants. We evaluated that by allowing in the model different CP growth during stabilization.

We also looked at improper institutional and consumer hot-holding. What would you - what would happen if you or what is the effect of abusive hot-holding on processed meat and poultry? What's the effect of improper cold storage during storage if you have the storage temperature too high or if refrigeration fails?

So these were some of the risk factors that are actually included in the modeling, and we can pull out the effect of these independently of one another and also their interactions from the model.

So for example if we look at illnesses due entirely to growth during stabilization, that is you get growth during, in the modeling at least, you get growth during stabilization but subsequently the food is handled correctly and no further growth occurs, you would still get a few illnesses caused by RTE and partially cooked products.

We looked at the effect of changing the growth during stabilization, and this is how it varies. Up here now we've got illnesses per billion servings.

So a $\log_{10}$ - 1-$\log_{10}$ growth, a factor of ten growth, we estimate 79 illnesses per year in the U.S. due entirely to growth during stabilization only, and that can increase very rapidly as you go to higher growths.

It doesn't increase linearly with these things. It increases very exponentially essentially because this is an exponential style of growth along here.

So if you look at illnesses per billion servings up here, at 1-$\log_{10}$ we're way down we can barely estimate it. I have to simulate billions of servings to get a number here, and 2-$\log_{10}$ growth you're starting to be able to see I,t and 3-$\log_{10}$ it's getting substantial, and 3-1/2-$\log_{10}$ it's really going up.

So the model is estimating that at current - at 1-$\log_{10}$ growth the stabilization growth contributes pretty negligibly to total illnesses estimated .07 percent.

If we look at improper hot-holding, the

modeling that we've done that includes hot-holding estimates approximately four percent of illnesses due to improper hot-holding. We know in fact that this is an underestimate because in the risk modeling we didn't treat hot-holding adequately.

The model for each serving is an independent but hot-held servings you're going to have a lot of servings held together, and they'll cross-contaminate each other. So we're probably underestimating by a factor which is close to the average number of servings that are heated and mixed together during hot-holding.

Hot-holding in fact if you look at the epidemiology it's responsible for more than 90 percent of reported outbreaks, although these are typically from raw product of course.

Again these will be biased towards institutional hot-holding because that's where you're going to see the outbreaks that are detectable and reported. There's no estimate independent of - estimate of the role of hot-holding in RTE and PCF foods and they are - these foods are not so likely to be hot-held in either.

If we go and look at what's the effect of improper cold storage, this is where most of the

illnesses come from as predicted by the model. Temperatures and times during cold storage, there are occasions during cold storage that temperatures and times are long and high enough - high enough and long enough to result in substantial growth of *C. perfringens*.

These are based on refrigerated temperatures measured at retail and home in a FDA and Audit International survey in 1999 where some refrigerated temperatures were as high as 21 Centigrade, which is clearly a failure of refrigeration. and the times that we assumed here are based on the *Listeria monocytogenes* as well as the risk assessment and also modified a little bit some data a pilot questionnaire administered on a USDA hotline.

These times are quite adequate, certainly at temperatures of around 20 Centigrade to get huge growth of *C. perfringens* during storage. So most of this comes from the small fraction of storage temperatures which are high.

So the conclusions of the risk assessment are essentially that most of the risk for *C. perfringens* illnesses are - is not from food processing plants, not during stabilization at food

processing plants. The principle risky activity is probably institutions and consumers inadequate - holding the food at inadequate cold temperatures and also possibly at hot-holding.

That is really the principle output that we get. For the second risk management question, what's the relative growth of *C. botulinum* relative to *C. perfringens*, for each of those stabilization standards?

We analyzed the growth of *C. perfringens* and the growth of *C. botulinum* and the problem here is that the growth of *C. perfringens* is not predictive of the growth of *C. botulinum*.

There are ranges of temperatures, at low temperature and again at high temperature. These curves here are growth rate plotted against temperature in various conditions for *C. perfringens* and *C. botulinum*. We used these curves in the risk assessment for *C. perfringens*.

This is the one for *C. botulinum*, and you can see that the *C. botulinum* curve is somewhat different from the *C. perfringens* curves.

The problem is that there's a region of temperature, at high temperatures and another region at low temperatures where - at low temperatures *C.*

botulinum can grow where *C. perfringens* does not. At high temperatures there's a region of temperatures where *C. perfringens* grows very rapidly but *C. botulinum* doesn't.

So without further information you can't say what would be the growth of *C. botulinum* if you simply know what the growth of *C. perfringens* is. That's really the output of this risk assessment with respect to *C. botulinum* for that risk management question.

We also evaluated some what-if scenarios. A lot of the growth that the model is predicting of *C. perfringens* is occurring at relatively low temperatures, between 13 and 20 Centigrade.

It's possible that that doesn't happen in the real world. We don't have information on what really happens in real foods at these temperatures in real conditions of storage.

It's possible that there would be competition between *C. perfringens* with psychotropic spoilage organisms, that is bacteria that grow well at these lower temperatures.

To do a formal analysis of that would require a similar sort of risk assessment, a similar sort of analyses for all the other organisms and so it

was beyond our capabilities at the time.

So instead we did a preliminary approach to see what could be the effect and to do this, we made some assumptions. For example, suppose *C. perfringens* doesn't grow 50 percent or 100 percent of the time below 21.1 Centigrade because of the presence of spoilage organisms which overgrow it during storage and retail?

If you - so if you see what the effect of that is, well 50 percent knocks out just about half of them, and 100 percent knocks out almost all of those growing due to bad storage conditions, and we're just left with 7,900 estimate out of seven or eight percent left.

We've done similar sorts - well conclusions of that. The overall effect of this possibilities are lower the estimated number of illnesses due to *C. perfringens* and an increase in the relative contribution of illnesses from hot-holding which would be the majority of the remaining ones.

It wouldn't affect the number of illnesses attributable solely to growth during stabilization because that's already occurred before the storage. We did various other what-if scenarios in the risk assessment and you can read that this year.

The risk assessment was peer reviewed by five external peer reviewers, which I am now told were chosen this way. I was unaware of who was chosen at the time they were done.

The overall review of the risk assessment report I think was relatively positive, a lot of comments. I responded to 234 comments I think it was. But they're basically mostly very positive - the primary criticism was the limited data availability, and I agree entirely with that.

They pointed out areas where a greater clarification was needed in the text, and I tried to do that in the current document. They did not suggest any changes in methodology, and they did not locate any additional relevant data that we could use in the risk assessment.

So the report was updated and now incorporates these clarifications, and there was no change to the calculations or results as the results of the review. So we developed a model to determine the impact of public health - on public health of altering the current CP growth critical control limit.

Our current estimate is approximately 113,000 illnesses per year predicted to be caused by CP from consumption of RTE and PCF if growth is at the

current 1-$\log_{10}$ limit. There's no data basically to validate this estimate and we are - we have considerable uncertainty from our modeling efforts, at least a factor of two in that estimate.

From the risk assessment modeling, stabilization at food processing is not a significant source at 1-$\log_{10}$ growth during stabilization, although there are some illnesses associated with *C. perfringens* growing in this process as even 1-$\log_{10}$.

The majority of illnesses are associated with improper cold storage of ready-to-eat and partially cooked food and the external peer review that we did didn't result in changes in our risk estimates. And at that point, thank you. I'll stop and --

MODERATOR GOLDMAN: Thank you, Dr. Crouch, for that very excellent and straightforward presentation of the risk assessment on *Clostridium perfringens*.

We're at the point in the agenda now where we have ample time I think, 45 minutes on the schedule to hear your questions, take any comments that you may have and try to provide answers as we can.

I'll remind you that if you have a question or comment, if you'll come to one of the two

mikes that are set up in the middle isle and identify yourself and your affiliation so that we can get that information for our transcript. I think we're ready for questions and comments. Yes?

MS. SCOTT: Jenny Scott from the Food Products Association, a couple of questions for you. Given that you estimated it takes large numbers of *Clostridium perfringens* to result in illness, was your dose response model a non-threshold model?

DR. CROUCH: The dose response model is in fact a non - that we put in for individual strains is a non-threshold model. It really makes very little difference in fact what you put in for the dose response for an individual strain because most of the variation that occurs is between strains.

You've got - and also most of the illnesses are caused by very high doses. You - the - what we're seeing in the modeling is that if *C. perfringens* grows at all, it tends to grow hugely, almost to a stable state because there's enough time - permissive temperatures there's enough time for it to grow.

So you've got an almost off-on phenomenon. It's just a question is whether you've got a big enough food serving to get enough cells into you and

if that particular strain just happens to be sufficiently potent to cause illness.

So the answer to your question is yes, we use the non-threshold model, but it really makes very little difference if we put a threshold model in for individual strains, there would be very little difference.

MS. SCOTT: Your model does take into account that only about five percent of the strains were enterotoxin-positive?

DR. CROUCH: Yes. Yes. We explicitly look for the fraction of type A CPE plus strains and we have a fraction of those in the uncertainty amount.

MS. SCOTT: In these numbers of illnesses, 113,000 for 1-log growth and was it for 138,000 for 2-log and 183,000 for a 3-log, in the context of risk assessments this - these all seem to be in the same order of magnitude. Do you consider those differences significant?

DR. CROUCH: The differences are significant. The increase is significant. What the model is showing us is how things vary. The absolute number we are uncertain of, a ? we've got considerable uncertainty about, but how they vary as you vary the growth during stabilization is probably fairly good.

So does that answer your question sufficiently?

MS. SCOTT: Yes, thank you.

MODERATOR GOLDMAN: Thank you.

MR. TAORMINA: Hi, Peter Taormina from John Morrell & Company. I wanted to talk a little - ask you - first of all, I commend you on the volume of work you did and I think you addressed your objectives set before you.

I did want to ask you about growth as it relates to chilling or stabilization. I think it's referred to as G_c in the model if I'm not mistaken.

Why were only limited - it seemed like limited studies were used to estimate this parameter. I think there was one for cured beef, one for cured chicken, one for ground beef, and a couple of times you've mentioned that there's a limited amount of data out there.

It seems to me that there is a lot of data. It just may not fit what may be your criteria were for using the data, like one of the reviewers noted that some references and your response to them was that the cooling data that they generated in actual product using actual cooling curves wasn't useful because it didn't - it wasn't - it was an integrated effect of growth rates over a specific

cooling curve.

I was just wondering - I guess I'm asking a lot of questions at once, but is there a way to incorporate data that is generated in a dynamic system during the cooling phase of a specific cycle rather than using different static growth rates and static temperatures to estimate a dynamic cooling growth rate?

DR. CROUCH: Can I answer your questions one at a time? First of all, what did we use for growth rate? We used primarily three studies to estimate the shapes of the growth curve versus temperature because they were basically the - they were provided a very large amount of data, more detail than anything else available.

We look for other measurements, but these were basically then what was available. I then did a literature search for and found, I think it was 174 measurements of growth in the literature and took it down to all of them in the risk assessment.

I used those to estimate the variability that one would see between servings and strains and other situations. The main use of the three detailed studies was to get the temperature variation across, so that's your first question.

The second question is during stabilization we had insufficient data on industry practices to say what is actually done in industry and how much growth could we model what is done in industry to say how much growth actually occurs now in industry, and we have basically no information on that. There's just a few cooling curves published that may or may not be representative of industry practice.

We can't tell. I mean, these were basically lab studies. So there are - I think there's a couple of measurements of real practice. So instead of attempting that, we went with assuming a certain amount of growth during the stabilization step. So that's why we did what we did.

Now your question also was could you do an analysis of what happens? The answer to that is strictly speaking right now no. We still - we could do it, but we wouldn't be very certain about it because we still don't know precisely how growth changes as you change - if you've got a dynamic situation.

We can model that, but we don't know if we're doing the right modeling. There are some data now coming out that will allow to evaluate that. I

mean one has an idea of what's going, and one can certainly integrate up growth curves - growth rates over cooling curves and take account of the delay period and stuff like that.

We don't know if we're getting it right yet. So if you're given a cooling curve you could - you can do that, and there is information coming that will allow better estimates of that in future, I think.

MR. TAORMINA: Right.

DR. CROUCH: Does that answer the question?

MR. TAORMINA: Yes, I think so and one paper in particular that you - that I just was reminded of was - I think it's by Huang.

DR. CROUCH: Yes.

MR. TAORMINA: 2004 where he in fact looked at a dynamic ? the effects of a dynamic cooling curve and a growth rate.

DR. CROUCH: Yes. It's not - I have some reservations about that modeling. There's a recent paper out in 2005 that I just heard about yesterday that also looks at this problem as well.

Well, actually, there was some earlier - there was earlier papers which attempted to do the

dynamic modeling but using an approach that was sort of ad hoc. It may be correct. We just don't know.

MR. TAORMINA: Are there - I guess at this stage, is it possible we'll use - is there an opportunity to incorporate some of the more recent data published in 2004 like the one we just mentioned and also ones that pertain to the effects of salt and nitrite like Zaika for instance in 2004?

DR. CROUCH: There's certainly - it's certainly possible to do that, to incorporate those effects. We have incorporated the effect of salt, and I put some effect of nitrite in.

The model is set up in such a way that if you knew how growth varied in industry for example, if you knew the distribution of growth rates, you can put that into the model. It's set up in that way. So the opportunity is there. Whether it will be done is up to FSIS, of course, not me.

MR. TAORMINA: Okay.

DR. CROUCH: It - I don't know that - well it depends what question you want to ask whether it's going to be useful to do that.

MR. TAORMINA: So are you saying - I think - are the food categories that you would plug in, would that suffice? Is that a way to - I mean, as the

categories are outlined that you can plug into the model, do you feel comfortable for, say, a less than three percent salt-cured meat item, or do you think there's a - is there a need for more clarification for those types of products?

DR. CROUCH: I haven't actually examined that question. We can examine it with the model by looking at what fraction of estimated illnesses come from the various types of - those particular types of food. It's just a matter of selecting them out. So questions like that can be answered, but I haven't got an answer for you right at this moment.

MR. TAORMINA: Thank you.

MODERATOR GOLDMAN: This is a good time I think for me to interject that this is an example just now of the reason we're here. We have worked very hard as you've just heard on a risk assessment.

We are presenting it to you, but the whole purpose of today as we've said earlier is to hear your comments and to incorporate your comments and especially new data that's available to us and especially from the industry in fact into these risk assessments so that they are as good as they can be in terms of representing the particular problem that we're trying to model here.

So we do appreciate your comments and in particular, your question about incorporating new data. That is part of why we're here. Yes?

MR. WHITING: Okay, thank you. Richard Whiting from the Food and Drug Administration. A question sort of on clarification here, you said the major driving force in illnesses then was improper cold storage. I gather that's not - a cooling going into cold storage, it's sort of the long-time storage of the food at say the ten to 20 degrees refrigerators that are not operating at the temperatures we'd like them to be. Is that a correct interpretation?

DR. CROUCH: That is correct. The survey data was very surprising to me, the surveys on refrigerator temperatures. Some of them, clearly, were broken refrigerators, I think.

MR. WHITING: Yes, I mean we've always thought of *Clostridium perfringens* that the unique characteristic of this organism was its ability to grow very rapidly from 35 up to 50 degrees.

What you're saying the driving force in all of this is not that characteristic of the organism at all. It's just the plain old long-term temperature abuse during storage.

DR. CROUCH: That's what --

MR. WHITING: Just like *Listeria* or any of the other pathogens.

DR. CROUCH: That is what our modeling is suggesting, subject to the problem about overgrowth and things like that that we do not know about.

It may be that overgrowth occurs and prevents *C. perfringens* growing in that temperature range, in which case the major contribution would be hot-holding or something like that. But it would be much less than the numbers I was getting there.

MR. WHITING: Okay. Thank you.

MR. SEWARD: Skip Seward, the American Meat Institute. On your initial slides - and I'm looking for some clarification here - when you talked about the types of food products that were used to establish the serving sizes that people would consume, and you had ready-to-eat foods and partially cooked foods, and it looked to me like from - that they were emerged, if you will, as you went through the risk assessment process. First though, is that correct?

I mean, were they modeled separately - partially cooked foods versus ready-to-eat foods because they, based on what you said, they had different dynamics in terms of vegetative cells and spores?

DR. CROUCH: The distinction is in the serving. The serving from the CSFII survey we treated all the servings the same through the risk assessment, but certain servings were considered partially cooked versus ready-to-eat.

The partially cooked ones - the difference is in the initial step where what we assume about the heat step and the growth during stabilization. That's the only difference between them really in this evaluation.

Subsequently, they are treated the same because the same process is occurring in all of them, and then there's a distinction in what fraction of them get eaten hot, cold, and hot-held as well.

MR. SEWARD: Out of all of the products then that were represented in those 607, if I understood it correctly, how many of those products actually represent products which are produced by federally inspected meat and poultry plants here in the United States? Do you have a sense of how many of those are actually represent products as produced in a federally inspected plant?

DR. CROUCH: I cannot answer that question because we just don't know. I would guess most of them, but I really don't know because there's no

connection between the survey information and where the food was actually produced.

MR. SEWARD: Okay, thank you.

DR. CROUCH: If we - we tried looking for such information, didn't we, to try and figure out various aspects of this, but we couldn't find anything useful - anything useable.

MR. SEWARD: Are those 607 different types of products, I assume those are identified in the risk assessment?

DR. CROUCH: Yes, the - everything is - you've got the raw data that went into it basically included on the Web site. The 607 identified as different recipes in the CSFII so that's the extent of identification of them.

They may or may not be identified as particular products in the sense of somebody - you could identify them back to a manufacturer. We didn't try to do that because we didn't need to, but you have all that information available in the risk assessment and in the accompanying material.

MR. SEWARD: Thank you.

DR. CROUCH: Yes.

MS. SCOTT: Jenny Scott, Food Products Association, a couple more questions. Your initial

numbers of *Clostridium perfringens*, vegetative cells and spores, a lot of it came from the literature. Were there other sources as well?

DR. CROUCH: We got that information from surveys of raw meat basically. It's literature plus an FSIS survey that I don't believe has been published yet.

MS. SCOTT: So it's not the published FSIS baseline studies. This is a new study?

DR. CROUCH: The FSIS baseline studies that I think you're referring to were not used because they do not identify spores and they didn't explicitly identify - they didn't confirm *C. perfringens*.

MS. SCOTT: Okay.

DR. CROUCH: These are - can you remember the names of the ? it's Kalinowski et al, this one paper.

DR. GOLDEN: There was one paper as Dr. Crouch just mentioned, Kalinowski et al. That's a sample approximately 200 meat and poultry samples and identified a certain portion of them as being positive for *C. perfringens*, then there was a study - and they look for spores and confirmed *C. perfringens*, then there was a study by Taormina, et al - thank you.

They looked at a larger number of samples,

about 500 and also identified a fraction that was *C. perfringens* positive; however, they did not go on to confirm whether those were actually *C. perfringens* so they're *Clostridium* positive and not necessarily *Clostridium perfringens* positive.

The study which Dr. Crouch, the unpublished study, was a special study that FSIS completed in 2003 that looked at about 600 samples from raw ground beef and tried to identify the presence of spores of *C. perfringens,* and again a fraction was identified as positive.

Those *Clostridium* were confirmed as *C. perfringens*. Those were the three studies that were used to identify these levels.

MS. SCOTT: My other question - both of you in your presentations mentioned the one outbreak from a ready-to-eat product that was a turkey loaf.

I don't know if you mean to imply that the problem that resulted in that outbreak occurred at the manufacturing level where it was produced or if there's something that happened to it after that fact that *perfringens* spores remained in it from the processing facility subsequently grew out because of improper holding or improper cooling or whatever. Could you elaborate on that outbreak at all?

DR. GOLDEN: Yes. That outbreak occurred in 1997. It occurred in New York. It involved - 18 cases were confirmed. It was a firehouse where firemen live or reside.

I can - at this moment I apologize, I do not recall whether first of all that outbreak - there was information on contributing some factors and if there was, what were the contributing factors. I think I know, but I prefer to get back to you once I know for sure without speculating.

Additionally that outbreak did confirm that it was from a ready-to-eat product that was purchased at a retail establishment.

MS. SCOTT: But that doesn't necessarily mean that it wasn't something that the firemen did with the product that subsequently resulted in the outbreak.

DR. GOLDEN: Right absolutely.

MS. SCOTT: Thank you.

DR. CROUCH: That information is all public. It's in the outbreak literature.

MR. HUFFMAN: Randy Huffman, American Meat Institute Foundation. A quick clarification, at the beginning of your presentation, Dr. Crouch, and I think Dr. Taormina eluded it to earlier, but I just

didn't quite understand.

When looking at the CSFII survey data that - the food consumption data, you mentioned that meats with greater than three percent salt and nitrite-cured meats were excluded, and maybe I misunderstood you, but could you elaborate on that?

DR. CROUCH: Servings which were both - which contained nitrite and more than three percent salt were excluded because *C. perfringens* doesn't appear to grow under such circumstances. It seems to be suppressed. Is that clear? Is that what you asked, what you wanted?

MR. HUFFMAN: So nitrite containing products - can you elaborate a little bit on the impact of that to your analysis? I'm not sure I follow how that relates to the modeling that was done. If it - does it apply to nitrite containing products or not?

DR. CROUCH: Products - I'm sorry. Servings that contained a lot of food recipes that were cured had nitrites and more than three percent salt were excluded from the final set of servings that were modeled in the risk assessment.

They were excluded for the same reason that shelf stable and servings with more than 8

percent salt were excluded because they wouldn't support the growth of *C. perfringens* so if you put them in the model they would just give nothing. It would be pointless to carry them through. So does that answer your question?

MR. HUFFMAN: Yes, thank you.

MR. SEWARD: Skip Seward, American Meat Institute. Help me understand something because I'm not totally familiar with the use of the terminology. I think you said something like you used the *Salmonella* multipliers to predict the illnesses from *Clostridium perfringens* in ready-to-eat products to help arrive to the estimates of 113,000 per year at 1-log growth.

DR. CROUCH: No, that's incorrect.

MR. SEWARD: Oh, okay.

DR. CROUCH: You misheard, I think. What I was saying there was that Mead, in his 1999 paper, when estimating the roughly quarter million illnesses made those assumptions based on reported *C. perfringens*. I think it was 34 or something like that.

DR. GOLDEN: There were - during over a ten-year period from 1983 to 1992 Mead - excuse me - identified an average of about 600 illnesses. Then

due to the fact that there was no passive or active surveillance of *C. perfringens* he multiplied that number by a factor of ten.

Then to account for underreporting of *C. perfringens*, he then used a *Salmonella* multiplier and multiplied that by a factor - excuse me - so the 600 times 10 now also multiplied by a factor of 38 to come up with the quarter of a million estimated illnesses caused by *C. perfringens* in the United States annually.

DR. CROUCH: That was his estimates, but nothing like that was done in the risk assessment here.

MR. SEWARD: Okay, thank you for clarifying that. When - at the bottom of that slide, I think there was a statement that's saying there were no data to validate the model, but didn't we hear previously that there was approximately 1,000 cases of *C. perfringens* per year over that extended time period versus 113,000 per year?

DR. CROUCH: What we have - what we know about is what reported as outbreaks to the CDC. Now when you get diarrheal illness, I don't think you usually report to the CDC, and, besides, an outbreak is defined as being well - Neal - Dr. Golden gave the

definitions of an outbreak.

So you miss a very large number of diarrheal illnesses in the population in the reported outbreaks. In fact you miss the majority of them.

That's why Mead was applying those multipliers, to make an estimate of how many there really are as opposed to how many are reported to CDC. That's the difference.

What the risk assessment is doing is trying to estimate the total number of diarrheal illnesses so that they would - most of them would never get reported even if anybody thought about doing it.

MR. SEWARD: But based on that data then we - is it - is the - there would be over 110,000 cases per year that were not reported based on the difference between what was reported and what the risk assessment model predicted?

DR. CROUCH: Yes.

MR. SEWARD: Okay.

DR. CROUCH: You don't - you expect almost none of these to be reported because they're not reportable basically.

MR. SEWARD: Thank you.

MODERATOR GOLDMAN: Let me just make a

point here with Mr. Seward's comment and question because this has come up in previous discussions of our risk assessments.

Each risk assessment model does come up with an estimate of total illnesses, as this one did. We usually put that in some context by presenting the data from the Paul Mead and CDC paper from 1999.

There are very often some questions about the differences in the total estimate that we get in a risk assessment versus the estimate that Dr. Mead and his colleagues got in their estimate.

The answer - the short answer is that there are different assumptions and multipliers put into the two different models, and we are not trying to - when we produce a risk assessment, in our estimate, we're not trying at all to challenge the estimate that Dr. Mead came up with.

Really the important point of having an estimate at all is you have an anchor point for modeling which - what the essence of a risk assessment is and modeling the changes in those estimates based on an intervention that can be made along the point of production.

So really the focus is not on the estimate that we use as the anchor point but on the changes in

illnesses that occur when we change performance standards or processing standards like we're discussing today. So I just wanted to clarify that.

DR. GOLDEN: I would also like to add that Paul Mead's and colleagues estimates are based from all foods. Of course, in the risk assessment we're estimating illnesses from ready-to-eat and partially cooked which would obviously be a fraction of all foods.

MR. DORSA: Warren Dorsa with John Morrell. That really - what you just brought up is why we're here discussing this and what's important. The question is how will stabilization process in meats affect human illnesses?

So that anchor point is extremely critical in these risk assessments, and actually when you read some of these conclusions, it almost - it leads me to believe that stabilization caused 113,000 illnesses a year, or at least that's the guess.

Yet further on you - in one of the conclusions, few predicted illnesses are associated with stabilization at processing facilities. To me 113,000 and limited to no associated illnesses are very contradictory, and so there seems to be some contradiction in the risk assessment.

MODERATOR GOLDMAN: Dr. Crouch --

DR. CROUCH: If you could write comments to point out where you're getting misled, I'd appreciate that because then I could change it so that it wasn't.

MR. DORSA: Well, one has to do with 1-log growth during stabilization, 2-log and 3-log. By the time you get to 3-log you're in - you're approximating several million - quite a few more illnesses due to stabilization.

DR. GOLDEN: I think I can address that. In regards to the current stabilization performance standard which is of course the 1-log maximum growth where we predict 113,000 illnesses, that not only includes the role of stabilization, but it also includes the role of improper hot-holding and improper cold storage.

As Dr. Crouch mentioned, 93 percent of those are - of those predicted illnesses are from improper cold storage. If you isolate the role of stabilization which is going to be a fraction of those 113,000 illnesses, it comes out to be about 79 illnesses per year.

So that is where the statement that stabilization contributes to a minimal amount. That's

where that comes from, from the 79 predicted illnesses. The 113,000 again includes what happens after the product leaves the processing plant.

MR. DORSA: All right, thank you.

MS. SCOTT: Jenny Scott, Food Products Association. Thank you very much for that clarification because that gets at something that I was trying to get clear in my mind and I think Warren was also trying to address this.

Given that you need 10^6 or let's even say a minimum of 10^5 it sounds like *perfringens* to cause illness and we're starting out with very low numbers - initial numbers presumably.

I apologize. I haven't time to read a 350-page risk assessment that just came out Monday before this meeting, and we're looking at - well let's say we have 1- to 3-logs of growth of those initial numbers during the stabilization then clearly how much illness results from that, it really is a subsequent mishandling that is applied on top of that stabilization that results in the illnesses.

There are really very few illnesses that would result if those products were properly handled once they left the manufacturing facility, correct?

DR. CROUCH: That's one of the slides that

I showed.

MS. SCOTT: Yes.

DR. CROUCH: The actual number that are due - entirely due to growth during stabilization. It should be - I should point out that in most of those cases you don't get a very large number of cells in the serving. You don't get a very large number of vegetative cells in the servings.

As you say, you're starting with a small number. You're only getting a small - a relatively small growth during stabilization. So you get tens of thousands of cells maybe or thousands of cells.

We are estimating from what we know about the dose response curve that a few in a billion of that - a few in a million of those cases which goes back down to a few in a billion servings may cause illness.

MS. SCOTT: Yes.

DR. CROUCH: Even though there's only a - I mean, you don't have a very large dose, but occasionally even a small dose may cause illness either because it's just the probabilistic thing or you've got a very potent strain. Most of it would probably be from the possibility of a very potent strain.

MS. SCOTT: Yes. How much information is there in the literature that that is an actual likelihood?

I mean, I worked on *Clostridium perfringens* for my Master's, and it was pretty much a given that you needed five - 10^5, 10^6 cells per gram to make someone sick, and we were not aware of any strain that caused illness at significantly lower levels than that, maybe occasionally you see something that was 10^4 per gram.

DR. CROUCH: Well, it's quite clear that you get a very large strain variation over several orders of magnitude, several factors of ten. So some of what we have done is an assumption that there are more potent strains.

The ? yes, in a lab situation you're going to have to give 10^5 because you want a high probability of seeing something. We're talking about very low probabilities here - 10^{-9} per serving is very low, but remember there are 55 - we estimated about 55 by 10^9 servings per year, so it's a very low probability in a very large number of servings.

MS. SCOTT: Okay, just going back to the vision point and what you have clarified there about the role of stabilization and its contribution to

illnesses.

I think that maybe that will be an area for clarification, that you bring that out a little more strongly maybe than what we have seen out because it keeps - the focus seems to be on if you have 1-log growth during stabilization there are going to be 113,000 illnesses, and 2-logs you're going to get 138,000, and 3-logs you're going to get 183,000 whatever.

Yet that still is predicated on the fact that there has to be some subsequent mishandling after that that would result in those illnesses.

DR. CROUCH: Well, remember also that there's an interaction effect as well. We've been talking about the total, which is all effects and then concentrating on just the growth and stabilization.

There's an extra effect due to essentially to the combination of growth and stabilization and subsequent cold storage. So if there wasn't the growth and stabilization during stabilization there wouldn't be the illness because the subsequent mishandling wouldn't have done it. It wouldn't have an effect. Do you see what I mean?

So you've got a those due to stabilization alone, those due to cold - bad cold storage alone as

it were and those due to the combination. So cold storage has the - sorry - so it grows during stabilization. It has two effects.

One is direct growth during stabilization only which is the one that I picked out but then there's another set that I hadn't picked out that I could, growth during stabilization and growth during cold storage, and it needs both of them to give you the illness.

I mean that's a more difficult one to pick out because you've got to do it in the situation with did it grow there and there? I mean it's a combination effect which gets more complicated.

MS. SCOTT: Right, and I believe your model does take into account the fact that *perfringens* dies off at cold temperatures.

DR. CROUCH: That's right. That's in there as well.

MS. SCOTT: Okay. Thank you.

MR. TAORMINA: Peter Taormina with John Morrell. Just a couple more questions - you mentioned interaction, and I guess that pretty much answered my question which was going to be does G_c growth during cooling interact with all these other parameters. I guess the answer would be yes.

DR. CROUCH: Well, it does in that sense. You may have situations where if you didn't have growth during storage as well as subsequent growth then you wouldn't have got the illness; whereas, in order to get the illness you need both happening.

MR. TAORMINA: Okay. The other thing I had a question on was, and you brought out some of this in the risk assessment, you discussed it at length, the estimates you used for spore concentrations in meat not quite - I wasn't able to - I mean, reading through in limited time I wasn't able to really find where you actually came up with the parameter estimate for spore concentrations in the meat fraction and what percentage of those - I mean, and also taking into account that less than five - well around five percent actually turn out to be CPE positive.

I wonder if you can kind of elaborate on that?

DR. CROUCH: The spores - the number of spores and the number of spores in meat fraction come from the three papers we were discussing earlier, directly from those, from analysis of those studies.

So that gives you the total number of *C. perfringens* and then we simply applied what fraction

those are likely to be CPE positive type A based on other measurements of that fraction.

Is that clear enough? All right. It's all there. It's --

MR. TAORMINA: Right. Is there an actual number?

DR. CROUCH: It's a lot to take in, I agree, in a short period, but you have it.

MR. TAORMINA: Right. Those are actually a lot - was there an actual number of spores that were used as --

DR. CROUCH: Well it's a distribution.

MR. TAORMINA: Okay.

DR. CROUCH: Because what we have is an observation of, for example, the FSIS survey saw two cases - two out of 593 samples had one positive - sorry - had a single observation of spores. So there was one colony-forming unit in each of those two samples.

In the Kalinowski we've got - I forget exactly how many there were, but there was one case there was more than one colony-forming unit.

From these very limited information, we've got a distribution of how many colony-forming units per gram of raw meat, how it varies and how uncertain

we are, which is a lot, about that distribution.

MR. TAORMINA: So what was the upper confidence limit or what was the upper range?

DR. CROUCH: I would have to - you would have to give me an exact question about that because it's difficult to give you an exact answer without a precise question as to what you mean by an upper range.

There's - in theory there's no upper limit because these are - these were - I modeled it with continuous distributions but they get smaller very fast.

You're most likely to find one colony-forming unit and you're most likely find none but after that you're most likely to find one and then going up less likely to find two or more.

I think the maximum that Kalinowski saw was eight, wasn't it, eight colony-forming units in a sample? I've got the data here somewhere. It's in the risk assessment.

DR. GOLDEN: I think after they did their calculations to identify how many spores per gram it came out to be about 60 spores per gram.

DR. CROUCH: That was a rather complicated experiment to analyze because they didn't confirm all

the *Clostridium perfringens* that they saw.

MR. TAORMINA: Thank you.

DR. BOYLE: Hi, I'm Dale Boyle with the National Association of Federal Veterinarians, and I claim no expertise. I've got a couple of questions and maybe comments. It depends on how you listen to the words I guess.

It appeared from the presentation that there were a number of factors that could change the disease outcome. It seems like the method of polling is an important feature to emphasize and report. Reheating controls it seems like is another important feature that should be emphasized in the report.

I didn't hear a lot about cross-contamination but that seems to be also something of major concern that I would worry about especially during the final preparation stage.

The other thing that I didn't hear addressed that might be useful for the meat industry is source material. Is there - are there some ways that you can minimize the amount that's actually being introduced in the first place.

My guess is that some of the controls that are being put in place for other pathogens over the past few years has also done a considerable amount to

reduce the amount of contamination that's occurring in the end product. So it may be that we've got a moving target as far as the level of pathogen that may be in the prepared product.

DR. CROUCH: I thought I'd just mention that you mentioned cross-contamination, but that turns out in this *C. perfringens* that doesn't seem to be a major effect, certainly not in final preparation because by that time you've either got the vegetative cells there or not. It doesn't matter very much if you - to cross-contaminate you'd have to transfer quite large quantities of food.

Cross-contamination is important for the hot-holding where we did not take that into account and we explicitly say so. That has little effect on the estimates of the variation as you vary growth during stabilization.

MODERATOR GOLDMAN: Okay, are there any last questions or comments for the morning session? All right, if not we've done very well on our time. We have a break ? oh, one more. Now we're over time.

MR. DORSA: Sorry, and I'll make it quick. Just since you did look like a lot of variables as you should in a risk assessment, would it be of value to look at what effect different beginning point

illnesses would have on the total risk assessment since it is in fact - it's an estimate with potential problems in the estimation so would there be any value in a risk assessment to evaluate what - the what-ifs - what if in fact the estimate is an overestimate and it's actually a smaller number or even a larger number.

Would there be any value in that or should that be something that should be considered in the risk assessment?

DR. CROUCH: I'm sorry, I don't really understand the question. What we've done is estimate the uncertainty in numbers of illnesses. What question are you asking? You may be asking a risk policy question rather than a risk assessment question.

MR. DORSA: Right, right but for the policy makers they're going to use the estimated numbers that you've put out in this risk assessment or started with as - in other words if you have a 1-log stabilization or an increase in during stabilization, the estimated illness is 113,000.

You've developed that number of 113,000 from an original estimate. Would there be any value - and the original estimate is just that. It takes into

consideration actual reported cases --

DR. CROUCH: No, no, no, you are misunderstanding. The 113,000 doesn't depend in any way on the - an original estimate of anybody else or how many illnesses there are. It's based entirely on measurements of *C. perfringens* in food.

MR. DORSA: Okay, all right, thank you.

DR. CROUCH: You're asking a question which is really - the question you are asking is really a risk policy question and so I'm not addressing it here at all.

MR. DORSA: Okay, thank you.

DR. HUFFMAN: Time for one more? Huffman again with AMI. Back to the question that Jenny asked early on, and maybe I'm just slow and didn't understand your response, Dr. Crouch.

When we looked at the three estimates at 1-log, 2-log and 3-log growth, 113, 130 and 180, Jenny pointed out that those appear to be within an order of magnitude, and she asked if those are different.

Well, as I look at the graph in the executive summary, you have something that appears to be error bars around those estimates, and it would appear to me that those are not significantly different, yet you answered that they are. Could you

expand on that a bit?

DR. CROUCH: Yes. Essentially consider those error bars as moving the whole curve up and down, all at once. So you've got the increase - sorry, that way around for you - you've got the increase no matter where you are on those uncertainty bars, you move the whole curve at once.

So it always increases by one point. If you - those are uncertainties so if we're really here it's still goes up like that by a factor of 1.21 and 1.57. They're correlated uncertainties. You can't compare here and here by just those uncertainties.

You've got to take account of the correlation. They are 100 - almost 100 percent correlated so that if it went - if you are uncertain about this one, you are uncertain in the same way about this one. So if this one has gone up, this one has gone up by the same fraction. Does that explain it for you?

DR. HUFFMAN: Yes, thanks.

MODERATOR GOLDMAN: Okay, I see a pause. Let us thank our morning presenters who will not be on the dais after noon. We'll reconvene at one o'clock for the presentation on *Salmonella*.

(Whereupon, the above-entitled matter went

off the record at 11:38 a.m. and resumed at 1:06 p.m.)

DR. GOLDMAN: I think we're ready to resume our discussion and presentations for the afternoon and just quickly on the agenda we'll again have an introduction from the policy and regulatory perspective from Dr. Daniel Engeljohn followed by an introduction to the microbiology and public health context by Dr. Carl Schroeder.

Then the presentation of the risk assessment itself by Mr. Paoli and then there will be a break and then we'll come back and entertain questions and comments and then we'll wrap up after that. So if I could ask Dr. Engeljohn to introduce this next risk assessment, thank you.

DR. ENGELJOHN: I'll give you a little bit of what I'm going to talk about is background on the proposed rule for which this risk assessment is derived, the risk management questions regarding Salmonella and then a summary.

Background on the lethality policy, we - the agency issued a final rule on cooked meat patties, roast beef and cooked poultry in January of 1999.

In that regulation we identified prescribed time and time/temperature requirements for cooked meat patties and we provided a 6.5-log

reduction for Salmonella in roast beef product and a 7-log reduction of Salmonella for cooked poultry.

We followed that final rule up with a proposed rule that would cover all ready-to-eat products other than cooked meat patties, roast beef and cooked poultry with lethality performance standards.

In that proposal, we added a 6.5-log reduction for Salmonella for all ready-to-eat meat products so this incorporated the roast beef products, the meat patty products as well as all those other ready-to-eat meat products that were not formerly regulated.

We maintained a 7-log reduction of Salmonella for all ready-to-eat poultry products and added a 5-log reduction for E. Coli 0157:H7 for fermented beef products.

We received comments on this proposed rule. Many of the comments that we received identified that, based on the levels of pathogens in the products, that the performance may in fact be too restrictive under those circumstances.

The design of the lethality performance standards were based on longstanding industry practices. We used a worst-case scenario assumption

in terms of deriving our lethality criteria and we used expert opinion.

So we did not at that time have a risk assessment to base our decisions as to how we should design the performance standards.

As a consequence then we asked our risk assessors to look at this issue so that we could address the comments that we had received during the proposed rule to inform us as to how we would go forward with the rule making.

The primary question that was posed to the risk assessors was what would be the public health impact of alternative lethality standards of a 5-log reduction and 6.5 or 7-log reductions for Salmonella, the 7-log reduction being for those products that are containing poultry.

With that primary question then we did ask a number of secondary questions and I'm going to give them to you in they are contained in the Supplement Risk Assessment that's available on the Web site.

I want to walk through these so that you'll have an idea of what kind of questions we are at least anticipating to deal with in terms of formulating our final rule-making policy.

The second question then would be what

would the impact of lowering the lethality for Salmonella on the following: A, lethality of *Listeria monocytogenes* in ready-to-eat products and B, lethality of E. Coli 0157:H7 in ready-to-eat fermented products containing beef.

Number three, what is the effect on public health if the Salmonella lethality performance standards for roast beef is also lowered to 5.0 and this would be from the 6.5 that had previously been put in form of a final regulation.

Question number four was what effect would the use of an integrated lethality of 5-log reduction have on the reduction of E. Coli 0157:H7 and on Salmonella?

The fifth question was if the process for certain products does not achieve more than a 6-log reduction for Salmonella, what would be the effect of retaining these processes in setting the performance standards as that all ready achieved? This would be by industry.

The sixth question, can the effect of Salmonella incidents from varying lethalities be determined?

Number seven, what is the effect on public health if only roast beef, cooked meat patties, and

cooked poultry have codified performance standards while all other ready-to-eat products remain non-codified?

Number eight, what is the effect on public health if only the large plants are required to meet the performance standard, the same for small and the same for very small?

What is the effect on public health if implementation is staggered over five years, that is, large within one year, small within three years, and very small within five years?

Finally, what is the effect on the public health if the performance standard is designed to account for production volume instead of HACCP plant size?

These are the questions that we proposed to have answered through a risk assessment and now we'll hear how that was constructed.

DR. GOLDMAN: Thank you, Dr. Engeljohn. All right next we will hear, as I mentioned, an introduction and overview to the risk assessment by Dr. Carl Schroeder.

Dr. Schroeder currently serves as a risk analyst in the Food Safety and Inspection Service Office of Public Health Science for about the last

three years or so.

Prior to joining FSIS he served as a Faculty Research Associate in the Department of Nutrition and Food Science at the University of Maryland in College Park.

Most recently at FSIS he has been involved in preparing the FSIS draft risk assessments for *Salmonella enteritidis* in shell eggs and Salmonella species in liquid egg products. Dr. Schroeder?

DR. SCHROEDER: Thank you very much. Good afternoon and thanks to each of you for coming today to listen to our description of the risk assessment.

Before I begin, in addition to Greg Paoli, I'd like to make mention of two of his colleagues, Todd Ruthman and Emma Hartnett, both also of Decisionanalysis, Incorporated who co-authored the risk assessment.

While a lot of individuals within our group at the Office of Public Health Science assisted, I'd like to make specific mention of my colleague Dr. Heejeong Latimer who's seated up front here. Dr. Latimer was instrumental in helping us review the model.

The purpose of my remarks today are just to give a brief overview of Salmonella and

Salmonellosis to help place the risk assessment which Greg will discuss in context. I'll give a brief background. We'll talk about the microbiology of Salmonella, epidemiology of Salmonellosis and a summary.

FSIS has proposed regulations that would require processors to achieve a specified level of lethality in the processing of ready-to-eat meat and poultry products; therefore, the required lethality can be expected to influence the level of public health risk which is associated with consumption of RTE, meat, and poultry products.

The Salmonella species in ready-to-eat meat and poultry risk assessment is concerned with the link between various alternative values of the required lethalities that FSIS would put forth as they relate to the resulting level of public health risk.

These are a few characteristics of the Salmonella. They are gram-negative, rod-shaped in contrast to C. perfringens non-spore-forming bacteria. They are facultatively anaerobic. They can grow with or without oxygen.

They're mobile by means of flagellae. They have optimum growth temperatures at around body temperature, somewhere between 35 and 43 degrees C and

they grow optimally at near-neutral pH.

Based on the often-cited work of Paul Mead and his colleagues at the CDC, it's been estimated that foodborne Salmonellosis each year in the United States is responsible for approximately 1.3 million illnesses, 15,600 hospitalizations and 550 deaths.

The disease characteristics of Salmonellosis include diarrhea, fever, abdominal pain, cramps, vomiting, headache and nausea. The incubation period ranges anywhere from 8 to 72 hours and symptoms can last up to a week.

The severity of infection varies. Most cases of Salmonellosis are self-limiting; however, some can be fatal and fatalities in severe illness from Salmonellosis is most often observed in young children, the elderly, and others who may have compromised immune systems.

Those who suffer Salmonellosis may go on to develop reactive arthritis. About two or three percent of all persons with Salmonellosis do so and a variety of other sequelae including urethritis, conjunctivitis, weight loss, oral ulcers and pneumonia.

We have had to institute several recalls due to Salmonella in ready-to-eat meat and poultry.

These are just a few examples here and what you can see is that these recalls have been linked to Salmonella in a variety of ready-to-eat meat and poultry products.

You can learn more about these recalls and find others at the Web site that I give on the bottom of this slide.

Lastly to summarize, we know that foodborne Salmonellosis remains a public health threat. RTE meat and poultry products have been recalled due to Salmonella contamination. The slide that I showed you earlier is strictly recalls. We also have epidemiologic data indicating that Salmonella in ready-to-eat meat and poultry has been linked to outbreaks of foodborne illness.

Today's risk assessment is concerned with examining the link between various alternative values of required lethality and the resulting level of public health risk. Thank you very much.

DR. GOLDMAN: Thank you, Dr. Schroeder. Now we will turn our attention to the risk assessment itself and Mr. Greg Paoli who is the principle risk analyst for Decisionanalysis Risk Consultants will present this.

He has been practicing microbiological

risk assessment for over ten years. He holds Master's Degrees in Systems Design Engineering and a Bachelor's Degree in Electrical and Computer Engineering from the University of Waterloo.

Within the field of microbiological risk assessment, Greg has served on a panel of the National Academy of Sciences of the Institute of Medicine and has served on multiple expert panels convened by, among others, the World Health Organization and the Institute of Food Technologists.

He is currently serving as a member of the FAO/WHO Drafting Group developing guidelines for risk characterization in microbial risk assessment.

Please welcome Mr. Paoli as he discusses the risk assessment on Salmonella in ready-to-eat meat and poultry products.

MR. PAOLI: I'll just test first of all that you can hear me okay. Okay. Well thank you very much for the opportunity to present the risk assessment.

I realize that I stand between you and the afternoon coffee break so in recognition of that I'll be as quick as I can and as quick as the task allows.

I'll first talk about the scope of the risk assessment. This will give you an indication of

what is included in the model and provide important information to put the result in perspective. It's also important of course to understand what's not in the model.

The risk assessment will be reviewed then in two phases, a quick overview to provide a broad perspective on the risk assessment to give you an idea of the key stages in the risk assessment and then I'll review some key assumptions. This will by no means summarize every detail of the model but give you a flavor of some of the more important details to consider as you review the documentation.

I'll then provide you with a few summary slides of the risk assessment results and I'll describe the uncertainty in the findings which is also very important to truly understand the results.

I encourage those of you interested in a more complete understanding to read the report and to browse the model when it becomes available to you. First of all just to reiterate the first risk management question that was posed and essentially reading it again.

It's important to understand the risk assessment to understand this question and its impact on the scope of the model, particularly when we're

talking about that the proposed RTE rule has a minimum lethality performance standard of a 6.5-log reduction in meat for all categories.

What would be the public health impact of alternative lethality standards of 5 and 6.5- or 7-log reductions of Salmonella?

As Dr. Engeljohn mentioned, a number of other questions which were posed and are dealt with, many of the results of those - responses to those questions are contingent upon the response to this question and we're really only going to deal with this first question today.

Answering all of the questions would probably take us into probably next Monday or so. So the scope of the risk assessment is estimation of the number of cases of Salmonellosis resulting from Salmonella in contaminated raw materials that survive the lethality treatments that are applied to ready-to-eat meat and poultry products.

Okay. So we're focusing on a very specific pathway by which Salmonella may contaminate ready-to-eat products. We're only concerned here with the risk that stems from Salmonella that survive the lethality process.

Also the risk assessment addresses 16

product categories which I'll lay out for you in a few minutes. Equally important, I would like you to take notice of what the risk assessment does not include.

It does not include illnesses caused by other pathogens, for example, E. coli 0157:H7, *Campylobacter jejuni*, or *Listeria monocytogenes*, although these are of interest. This was primarily a technical limitation in the ability to do that - do the model and I'll explain that in a few minutes.

In addition, the process applied to kill Salmonella - oh sorry - the assessment also does not include the risk that stems from post-lethality product contamination, that is Salmonella which first contaminate the product after the lethal step in the process. Okay.

Further it does not address the risk associated with what I'm calling an acute process failure where, for instance, such as might happen when there is a problem with the natural gas supply during the cooking process.

For instance, the rational for excluding these pathways of exposure is that though they may be important, they are not impacted by the level of the lethality standard.

For example, a fuel supply failure or some

other cause of an acute process failure does not become more or less likely as you adjust the performance standard.

The next slide here gives you essentially a conceptual view of the scope of the risk assessment. The box that you see on the left of the slide essentially shows you the scope of the risk assessment.

As you turn the dial at the top left so these are your five, six or 7-log lethality standards, you will have no impact on the risk which may be associated with contamination or failure of - outside of the box, okay.

So that, what's outside of the box could also include any Salmonella which may contaminate the product after including all the way to cross-contamination from other foods in any number of events down the process.

So what are the issues associated with that particular scope? One is that validation is not - validation data particularly applicable to this pathway, this particular scope are not available.

Data that describe the contamination of ready-to-eat product which would be clearly something they could use to validate, would however include

post-process contamination as well as any other cause such as an acute process failure.

So we can't directly use that, although it might constitute an upper bound on this situation. I'll discuss the capacity to validate the assessment considering public health data later on in the discussion.

The choice of product categories is another question. It's largely a matter of making the analysis feasible. We do not consider each and every ready-to-eat meat and poultry product and as many of you will know the diversity in these products is enormous.

So the categorization process is really applied to make the analysis tractable. It constitutes in itself the categorization a source of uncertainty in the model.

We do consider however the most important in high-volume products. The product category span the four processes that are of concern, thermal heat treatment, fermentation, drying and salt-curing, recognizing that for some products these may be to a certain extent combined.

This slide is intended to provide you with a conceptual view of the risk assessment. So here you

can imagine that there are six sliders designated by these little diamonds in which - for which you can use to control the level of risk in a product.

In each case as you move to the left, the risk is decreased and as you move to the right, it is increased. The movement of each slider moves the arrow indicator at the bottom along the risk scale at the bottom and not all the sliders will have the same impact on the risk.

What I'll be describing to you in the next few minutes is the process of placing each of these product categories along each of these continua so that we can come up with a product risk indicator at the bottom. I'll describe the risk assessment process in the overview sense as having five stages.

Stage one incorporates these tasks. One is develop - to develop representative product categories. Having assigned those product categories we assign raw material streams to those product categories and then we estimate the expected number of organisms in the raw materials for a given mass of products.

In stage two of the risk estimation process, we apply the lethality treatment at the prescribed level, that being 5 or 6.5 or 7-logs as the

case may be under a number of scenarios.

We adjust the lethality treatment based on compliance and this compliance data I will describe later and we apply thermal process safety factors which may apply and again I will describe those in some detail later.

Thus at the end of stage two we have an estimate of the number of surviving organisms in a given mass of product. In stage three we estimate the growth of the organism population during storage at retail and in home, if any, understanding that some products will not allow any growth.

We will apply heat treatment by the preparer, if any. So this is the heat treatment applied just before consumption, not to be confused with the process, the lethality process in the production of the product.

Thus, having done this, this provides a distribution of the number of consumed organisms in servings. In stage four we apply a dose response relationship to convert the distribution of ingested doses, number of organisms consumed into the probability of illness.

So this provides us an estimate of the expected number of cases of Salmonellosis for a given

mass of product.

In stage five we apply the amount of consumption of each product category in a year and then this is simply a multiplication which provides an estimate of the expected number of case of Salmonellosis in a year for each product category and then in total across all of the product categories.

I'll just quickly describe to you how the model was implemented. It was implemented using some modeling software called Analytica. One of the benefits of this as far as you may be concerned is that a player version of model is available which allows you to browse and run the model.

That - I'm not sure what the availability - when the availability of that will be, but my understanding is that it will be made available.

The next slide is just an example of what you will see if you download the model so that you have essentially a user interface. There's a great deal of transparency in that anyone can simply change the assumptions that have been made as the baseline to see what the impact is and various buttons you can click on to get the results, very much like the results you see in the report.

Just an example of one of the modules

described in growth and its ability to provide you with some numerical and graphical results. Okay. I've just come back to this conceptual model just as a reminder to you. What I'm going to be going through essentially is I'm going to be going from the top to the bottom of this slide and describing what's taken into account in assigning the products to different points in these continua and how it all works together.

I'll now go through and review key assumptions and the assumptions across a number of different areas that you may not be used to considering as assumptions.

One for example is the designation of product categories. Designing - grouping products together necessarily requires some problems in estimation.

Ideally we would consider each and every ready-to-eat meat and product on its own with respect to its particular parameters. I think you'd agree that that's not tractable and if you know a way of doing it in a reasonable amount of time I'd certainly love to hear about it.

Data selection and treatment - how do we treat certain data that we do have is clearly an area

of assumption. The estimation methods in any simplifications that we apply also are important to consider and also the issue of just reasoned assumptions in the presence of data and theory gaps that we have to - we simply have to make where we don't have any evidence to go on.

So the designation of product categories is an example of an assumption. This is essentially a design in the risk assessment. It's a compromise between a number of competing requirements.

One is that it be compatible with the risk management questions and that it addresses categories as they are known and regulated by FSIS.

They also should be compatible with data sources. So sometimes we group things together because there is data available which groups these things together.

We also need to make distinctions that are important to the risk estimation process in assigning things to categories. We, of course, need to have a manageable number of categories.

We cover major products in all four of the lethality categories - cooked, fermented, dried and salt-cured. I think I went up there. Okay. These are the product categories. The top - going from the

top all the way down to poultry frankfurters which is about 2/3 of the way down are all cooked products.

We then have some fermented and direct acidified products, some dried products, and some salt-cured products at the bottom. Again, these are essentially representative products and there are obviously some that span multiple categories. There are some which may slip through the cracks but this covers a whole lot of product.

I'll now talk a little bit about the assumptions under the - what I call the raw material pathogen burden, which is essentially a number of organisms in the raw material that we need to address with the lethality process.

This is based on the FSIS Microbiological Baseline Surveys primarily because number one, they are consistent across all of the products and they also provide the very necessary piece of data which is the level - the number of organisms, as opposed to simply the prevalence and that's a key requirement for this particular risk assessment.

We estimate the expected number of Salmonella in a given mass of raw materials, and this can be expressed on a per gram or per million kilogram basis.

We make separate estimates for beef, pork, chicken, and turkey and we also do this for both ground and intact versions of each of these.

This slide provides you with some indication of the relative pathogen burden. This is basically a merger, to put it crudely, a merger of the prevalence of the data as well as the concentration in - that was found in the baseline surveys, essentially coming up with a weighted average of the contamination levels in the product.

Given that we have 16 product categories, you'll see that I'm not going to go into a whole lot of detail on any one of these risk - these assumptions. You'll certainly be able to see it in the report.

I'm just going to do a quick summary of lethality treatments, although I think with the - looking around the room at the people that - who are here this, may not be necessary but it's the base 10-logarithm of the reduction factor.

Essentially it's a 5-log reduction means the population will be reduced by on average five factors of ten or 100,000. Equivalently we could say that each organism has a one in 100,000 chance of survival of the process.

If a million organisms or 6-logs were subjected to a 5-log process, we would expect on average ten survivors. So we go from 6-logs being a million down to 1-log with a 5-log reduction. Excuse me.

Three alternate policy scenarios were requested of the risk assessment and these I'll describe essentially with these labels. All 5-log means that all products require at least a 5-log reduction.

All 6.5 or 7-log implies that all products require a 6.5-log reduction, except where they contain poultry where they require a 7-log reduction.

A split scenario is what you might call the default scenario in the sense that where if it's not otherwise stated in the document this is the scenario that's described.

All cooked products require a 6.5- or 7-log reduction and all other products require a 5-log reduction, one exception being fully cooked beef patties which would require a 5-log reduction as is the current requirement.

A 1-log - yes, I think I explained that all ready. Okay. So this just gives you a visual indication of how that process works. The all 5-log

scenario is quite simple. It's the left most column of numbers. Everything requires a 5-log. Similarly the right-most column of numbers provides 6.5-log reductions for all categories except the ones containing poultry.

The split scenario is in the middle and it's a bit more complicated. You see cooked products receive a 6.5- or 7-log reduction as they did in they all 6.5- or 7- log scenario, with the exception of the 5-logs for fully cooked meat patties.

All non-cooked products that - and that is essentially here all of the products below the line that you see going across the table would require a 5-log reduction.

The next stage in the process is lethality compliance factors and this was based on an expert elicitation study done by RTI published in 2004, published -- I mean for the purpose of being complete, I don't think it was published in the peer view literature.

What proportion of the producers of product Y achieve an X-log reduction? Among many other questions that were asked, this is the one that's of interest to us.

So for instance, what proportion of the

producers of roast beef would achieve a 6.5-log reduction? For purposes of the risk assessment, full compliance results in some additional lethality being assumed.

Essentially what this is is an assumption that in being in compliance with the standard, some overshoot is generally designed in or some margin is designed in to make sure that you're not just in compliance half the time, which I don't think is what people would like to be.

It assumes based on this expert elicitation study that all cooked products are in full compliance with the 6.5- or 7-log reduction as required.

Deviations from full compliance however result in a reduced lethality so even though the scenario suggests a 6.5-log reduction, if the compliance is not there the net effect of lethality is weighted according to the level of compliance suggested in the expert elicitation study.

This is also a factor which can be very simply removed from the model, such that we assume that everybody achieves exactly the scenario or the standard that has been requested in the scenario.

I'm now going to talk about thermal

process safety factors, which is one of the more challenging - most challenging parts of this risk assessment and it really relates to the family of cooked products.

Essentially here for a variety of reasons, processors may apply a process that yields a much smaller average probability of survival that is implied by the strict interpretation of being in compliance with the required lethality.

So an example, in complying with the 7-log reduction requirement, a process may actually achieve a mean probability of survival that's equivalent to an 11-log reduction. Much smaller numbers and much larger numbers are very easy to contemplate and even give examples of.

Some of the reasons for these safety factors is - are essentially come down to things like the product geometry and the fact that we're heating all the way to the interior of a massive product and therefore there's a lot of heat - a lot of higher heat treatment being applied to the outside of the product.

Gradually as you get to the middle, the heat transfer to the middle is what - excuse me - what ultimately creates a much larger net log reduction than is actually implied by saying that they are in

compliance.

Another thing would be simply a consumer preference or quality requirements that the product be cooked to a certain level, which may result in a cooking that is much higher than is required by the standard.

Another reason why these safety factors come into play, and we did some calculations on this front which were somewhat surprising, simply the design and validation of processes with strains that are much, much more resistant than average and this seems to be very common, at least as far as is reflected in the literature.

For instance to - if you include in your validation step a cocktail which includes Salmonella enterica serovar Senftenberg which I have trouble saying after three years of this, this results in a much, much higher log reduction than is implied by the - really the rest of the cocktail.

In order to - basically in order to kill this bug you have to create a much, much more lethal process than you would otherwise. This organism is by far, it's a real outlier in this game.

So another reason might be where contamination of the product is limited to the surface

of the product, combined with the intense heating to warm the inside results in essentially a very high net reduction of the number of pathogens.

Now the estimation challenge here and this is what makes me want to go back to electrical engineering is that we know that these safety factors exist and that they can have a very large impact on the risk estimation process, particularly for certain products.

They may be simulated or known for certain products and processes so it - the calculation at the level of an individual process is entirely possible to calculate this net reduction.

What we need to know is the net impact across a whole industry because that is what the question asks. It doesn't ask a particular well-characterized process.

Another competing factor of this is that the industry-wide thermal process safety factor if - when you do the math behind it, it's strongly influenced by the proportion of the processors which for whatever reasons have relatively low safety factors.

This is very similar to what you heard about the C. perfringens is the domination that you

see by the extreme scenarios. In this case it's the domination of the risk by those having relatively low safety factors, even despite still being in compliance.

Another estimate - part of the estimation challenge is that most of the data is geared towards assuring compliance. It's not readily applied for estimation of product risk. So the risk that is being managed is the risk of being out of compliance as opposed to the risk that we're trying to estimate which is proportional to public health risk.

So this requires reasoned assumptions and what I'm saying here is that there is no fundamental way to do this other than to make certain judgments. This is implemented as having three possibilities.

One is to - as having a small safety factor which is essentially no change relative to the lethality standard, a medium safety factor which is a 2-log additional increment to the lethality and a large safety factor which would be a 4-log movement.

Each product category is assigned to one of these three factors. The model allows adjustment or in fact given the challenge associated with this and the potential discomfort associated with these parameters simply it can be removed from the analysis,

with the caveat there that the removal of the thermal process safety factor will virtually guarantee an overestimate of risk, particularly for the cooked products.

Okay. I'll now talk about this survival - single organism assumption which is really required to understand how we go from figuring out how many survivors there will be to what the risk is from the product overall.

The assumption is that survival of organisms is modeled as a rare event with respect to individual serving sized pieces of ready-to-eat product.

These rare events only become appreciable when we consider the very large number of these servings that are consumed each year.

Furthermore, not only are they rare, we would expect and assume in the model that these rare servings that do remain contaminated would be - would have only one surviving organism.

This situation and this is prior to growth, I should say. This situation has implications for our ability to validate by observing outbreaks. So from this particular pathway of contamination where it's simply a result of survival of a lethality

process. At the one in a million level we would expect that the resulting illnesses would be rare and randomly distributed according to where those particular finished products end up.

We would not expect to observe them as any kind of an outbreak. The opposite would be true for a process failure event or a significant level of process contamination where there would be significant clustering due to the causality of the event.

So we don't expect to see a bunch of outbreaks resulting from survival of lethality - lethal processes. Okay. The next area is storage and growth and here, given the incredible diversity of the products that we're talking about, we have assigned them to different - to four different scenarios.

One is no growth, where this is the product simply does not allow growth regardless of temperature. We have a category of low survival which is where we would expect a further 1-log reduction during storage and this 1-log is somewhat arbitrarily chosen because, again, we're categorizing this across an incredibly diverse number of products.

So we have to apply a number to give that indication of what the risk reduction associated with this process. Another category is normal growth -

excuse me- under refrigerated storage and here we're only talking about growth where the temperature would allow it. We're not assuming that it's going to grow all of the time.

It's going to grow where it - in situations where it's held at temperatures that would allow the growth of Salmonella. Similarly we have a low-growth scenario and here we just have half the growth rate of normal growth with the minimum temperature applied.

Thank you. Why do we go down this road? Well detailed growth modeling for this diversity of products even within one of these product categories would be a considerable challenge in itself. The data in the models required to accommodate this variety in distinct products are relatively limited compared to the diversity, although that situation is improving considerably as time goes on here.

I won't bore you with the details of the square root model, you'll be happy to know. For products that do allow growth, we model the growth in two stages, retail and consumer storage and a variety of time and temperature distributions are provided for in the model but we have carried through a certain default which you'll - time doesn't allow me to go

into. There are a number of scenarios available.

The next phase of the assessment is to describe the impact of reheating. Here again we have three levels of lethality which we could have due to rehearing. Again, we're considering a very diverse number of products and very diverse consumer practices associated with these products.

So one extreme would be no reheating whatsoever. At the other extreme is a thorough reheating which is a 4-log additional reduction. Again, this reduction may be applied to a scenario - a population that has occurred after growth.

In other cases it's happening as a 4-log additional reduction in a product that doesn't allow growth. The products are assigned to reheating pattern categories because it's not really ever true to say that a product is -- always receives a 2-log reduction or always receives a 4-log.

It's essentially a pattern of consumer behavior ranging from never, rarely, usually, always, and always thoroughly. These correspond to alternate patterns of assigning these different levels of reduction so always thoroughly clearly you can imagine is - places most of the weight on the 4-log additional reduction.

The category never means that all products get no additional reduction. The others are obviously in between along the continuum.

The next phase in the estimation is to apply a dose response model and we have adopted the Beta-Poisson model that's based on outbreak data and was developed in WHO and FAO expert consultations. Excuse me again.

This converts the dose of organisms which in some - in many cases will be single organism and in other cases a distribution of organisms resulting from variations in growth.

It converts this into a probability of illness. Note here that there is no minimum infective dose applied and that's consistent with the WHO/FAO Hazard Characterization Guidelines from an expert consultation there.

As is normally - as is applied in this dose response model, the probability of illness from a single Salmonella is about 2.5 chances in 1,000. This just gives you an idea of that dose response relationship.

If you look for instance along the X-axis you'll see for a 4-log dose or 10,000 organisms, you'll have approximately a 50 percent chance of

illness and then there's some uncertainty around that, which is considerably larger than what's actually shown there.

The next stage is consumption volumes and in the case of this risk assessment because it was dealing with a slightly different set of questions, we were able to use the economic census data because of the relative compatibility with some of these product categories.

For a few product categories it was based on a database product containing this CSFII data which was described earlier today. There's a lot of uncertainty, particularly for smaller volume products because they are not represented well either in the census or in the CSFII data.

Essentially they become relatively rare servings in the CSFII data and they would become relatively less important in the process of economic census.

I'll next - I'll now proceed to talk to you a little bit about the risk estimates which are - come from this proces and I'll talk to you a little bit after that about the uncertainty which is quite considerable.

We end up in a similar situation as was

described for the CP risk assessment where the tool is best applied in reasoning about lethality as opposed to assigning an absolute level to the number of illnesses.

Again I want to go through two different measures. One is the annual cases per million kilograms product class. This gives you essentially an equal mass risk estimate and then I'll give you the total number of cases due to the product class which considers the consumption volume because this has a considerable impact in this one variable, obviously, how much of it is eaten.

Then the model I won't be able to go through all of these options here but the model itself allows one to exclude the thermal process safety factor to exclude the process - the consumer reheating, exclude compliance adjustments.

Those are just examples. You can exclude pretty much whatever you want in the model. Okay. I won't expect you to be able to read this so having reviewed the model inputs in the form of data and assumptions, we now proceed to review the risk estimates.

I'll describe the slide for you. It's somewhat crowded but it's hard to place 16 product

categories on the same graph any other way. You'll now be glad that we didn't use 32 or 64 product categories.

The first thing to note is that the risk assessments - risk estimates are provided on a log arithmetic scale. This is out of necessity because the risk estimate span a range of a million, which is hard to graph any other way than on a logarithmic scale.

To help you with the log scale, the vertical lines designate differences of a factor of ten so as you go to the right you increase in risk by a factor of ten for each vertical line that you're crossing.

The point at which the bars meet in the middle constitutes one illness so that's essentially log-0. So this constitutes zero and this constitutes 10 and 100 and so on.

This particular graph is on an equal mass basis so this is the number of cases from a million kilograms of product. I'm going to focus on three product categories purely for illustrative purposes so that you can follow the product category risk estimates through a number of slides.

It's not intended to pick on these

products in any particular way. It's purely for illustration. One of them is cooked chicken, non-deli products here. Salami which is SUP here and meat sticks which is MS right here.

Okay. Keeping in mind that these are on an equal mass basis you'll see that fermented and dried products respectively have the highest level of risk on an equal mass basis.

On the next slide you'll see the impact of consumption volume which significantly alters the risk profile at the level of the population health risk.

Cooked chicken has a comparatively low risk at the equal mass level but becomes more important when you consider the sheer consumption volume involved.

This slide gives you an idea of the annual product risk and again we're on the log scale and now we're weighted by production.

So this is again the number of cases per year estimated in the model and once again focusing, you see now that cooked chicken has come up in relative importance due to the sheer amount of production of the product. It has now become comparable to the meat sticks and the salami products here.

Also note that as I referred to earlier this is under the split lethality scenario where you have 6.5- or 7-logs for the cooked product and 5-logs for all the uncooked products.

Another thing I want you to realize here is the - when I talk about the uncertainty later, when I talk about uncertainty with respect to factors of ten you can imagine that the error bars around these cross a number of these factor of ten lines. That's simply a reality of the estimation process.

I'm now going to go through a few slides for the three different lethality standard scenarios that I provided earlier, this is the all 5-log scenario.

The next two slides after this will show the split scenario and then the all 6.5- or 7-log scenario and the progression of the slides corresponds to increasing stringency in the required lethality.

The number at the top is the best estimate of the number of cases per year across all the products. The pie charts give an indication of the breakdown in those cases by product category.

So I'll just sort of walk you through this quickly. The other category here is further broken down because it would be impossible to show some of

these very small risks on the same pie chart. So this break down here is a break down of the other ten percent, okay, just to make that clear.

Here you see under the all 5-log scenario which is not the - which is not what is currently believed to be the case because you'll recall that I said that the current expert elicitation suggested that we were - we had compliance at the 6.5- or 7-log scenario for all of the cooked products so this is just a scenario which is not the current scenario.

Most - the risk is dominated by cooked chicken under this scenario. For instance, the meat sticks and the salami product are relatively small in this scenario.

When we go to the split scenario, what's changed here is that the cooked products have gone from a 5-log scenario to a 6.5- or a 7-log scenario. That significantly reduces their - the risk associated with the cooked products in this scenario as recalling that going from 5 to 6.5-logs is a factor of 30 reduction in risk at the simplest level.

Note now that we have a significant reduction in the best estimate of the number of cases. We've gone down to 1,900 cases per year. Fermented products and dried products now make a comparably

larger contribution to the overall risk relative to the previous scenario.

This is due primarily to the reduction in the contribution of cooked products. So essentially we have pushed down the cooked products and now we're on a more diversified product risk scenario I guess you might say.

The last scenario is where all products require an all 6.5 or 7-log reduction. Here the estimate is reduced a little bit further to 1,100 cases per year but not nearly as much as before. Recalling, we went down significantly between the first scenario and the second scenario.

So relative to the previous slide, this constitutes a more stringent standard applied to fermented, dried, and cured products. In this scenario, the fermented and dried products have decreased in their contribution to risk even more.

I'll now talk for a few minutes about the uncertainty in the model which is quite considerable. I think it's considerable in all of the risk assessments that are produced but I find this one to be one of the higher levels of uncertainty that I've come across.

I've provided you a list of the major

sources of that uncertainty and I can go on and on about what the rationale and why we're so uncertain but in reality the number of stars that you see after is somewhat of a qualitative indicator of how uncertain these things are.

Obviously the thermal process safety factors that I applied, I spent some time telling you how - that they're very important, that they're very real but we don't have a concrete way of measuring them at this point.

The raw material pathogen burden is relatively uncertain. The dose response is also uncertain because for many of these products we're assuming exactly one ingested organism in the contaminated serving. So we're very, very reliant on that particular estimate.

As I said I could go on and on about the uncertainty but the model - ultimately we have to understand that the risk estimates presented should be considered to fall within a broad range of uncertainty. They may be several factors of ten, smaller or larger and whether they're likely to be smaller or larger depends on the product category.

Given this uncertainty, the relative rankings or the attribution of total risk should be

considered correspondingly uncertain. In summary and I have no idea where I am in terms of time. I'm okay?

DR. GOLDMAN: You're ahead of time.

MR. PAOLI: You haven't dragged me off or anything. So the risk assessment provides policymakers with estimates of the impact of alternate lethality standards - 5-, 6.5- or 7-log reductions or other ones actually. There's no reason they can't be put it in terms of the software product that was created.

On the expected number of cases of Salmonellosis there are 16 product categories. The software is designed to allow for exploration of the impact of alternate assumptions at numerous stages in the estimation process.

The model in the report will be revised in response to public comment. Thank you for your attention at this very difficult time of day.

DR. GOLDMAN: All right, thank you, Mr. Paoli. We are at our afternoon break so we have scheduled a 15-minute break and then we'll come back and take your questions and answers and then we'll wrap up. So we'll be back at 2:20.

(Whereupon, the above-entitled matter went off the record at 2:05 p.m. and resumed at 2:29 p.m.)

DR. GOLDMAN: I think we're about ready for the question and answer and comment period.

Do we have any questions or comments for our presenters from the afternoon session?

MS. SCOTT: Jenny Scott, Food Products Association. Greg, would you clarify for me what baseline studies were used for the Salmonella data and what time period they cover?

MR. PAOLI: They - the baseline studies where the ones ranging in the `94 through `98 range - I can't remember the dates off my head but essentially the ones that are on the Web site now as the FSIS baseline studies.

MS. SCOTT: Do you have any more current data?

MR. PAOLI: There is more current data but not more current data with respect to levels of organisms. That's the crux of the matter and when I indicate a fairly high level of uncertainty associated with the raw material pathogen burden.

Although I didn't get into the details of that, that uncertainty is primarily whether that survey of a decade ago or however long it is now constitutes an adequate representation of the current state of the raw materials.

MS. SCOTT: Right, and it would be my concern because we know prevalence has gone down significantly. I would probably suspect that numbers have also gone down as well but without doing more testing we can't be sure of that. I just wanted to see if there were any other sources of data that would be a little more current than what was in there. Thank you.

MR. PAOLI: Yes.

DR. GOLDMAN: Other questions or comments?

MR. POWELL: Mark Powell, USDA Office of Risk Assessment and Cost/Benefit Analysis. Just one comment I think just to emphasize one commonality I think between both assessments that I think all of the analysts involved are well aware of but maybe we need to remind ourselves of is that these are both looking at indicator organisms.

Perfringens was selected as the indicator organism for a sweep of similar pathogens of concern similarly. Salmonella was chosen as an indicator organism for lethality standards. There's ancillary effects of lethality and of rapid stabilization of ready-to-eat products that can't be captured for lack of available information.

Both - excuse me - both analysts referred

to that but I think we need to simply remind ourselves of that.

DR. GOLDMAN: Okay, thank you. Any other questions or comments? Well done, Greg and Carl. Well, if you think of a question as we move forward then you can still ask it but hearing no other questions or comments right at the moment.

I think we will then proceed to wrap up what we've heard today and another thing I'm sure you're interested in is what the agency will do next with respect to these two risk assessments and its policy decisions. So for that I'll ask Dr. Engeljohn again to begin the wrapping up.

DR. ENGELJOHN: Thank you. The next stage is then for the risk management perspective with regards to where we go as to how the risk assessors address the comments that we receive, both today as well as throughout the next 45 days.

The comment period will end on May the 9th so be sure that you take the time to read through the complete risk assessments. For those of you here in Washington, all the support documentation is available in the docket room for your review as well. The comment period will remain open until May 9, which is a 45-day comment period.

At the same time the risk managers within the agency will begin analyzing the responses that we have thus far from these two risk assessments and from the questions that we posed and try to make some decisions about how to go forward with the rulemaking that we proposed back in January of 2001.

It was the agency's intention to go forward with rulemaking and for those of you who may remember that rule, the rule was one in which it included the Listeria component which we did in fact finalize in October of 2003.

It also contained a section that dealt with thermally processed products, the canned products as well as a Trichina proposal. So those issues would be taken under consideration as well as we go forward.

In any case, the risk managers now will be looking at the information that we've gleaned from these two risk assessment. We will begin the process of looking at the economic impact of any of the decisions that we made.

There were some contracted studies that the agency did to get more information about what the industry was capable of doing and what we thought they actually were doing with regards to their control procedures.

That information will also be available in the docket room and will be used to help us make some decisions about any impact that would occur with the decisions that we make from a risk management perspective.

I think also from - particularly from the Clostridium perfringens risk assessment, it's clear that much of what needs to be done does in fact need to happen outside of the federally or state inspected facilities and it would be greatly impacted by what happens by the retail co-chain distribution as well as the consumers.

For that end, the agency needs to be looking at what it needs to do for outreach and education with regards to proper handling of product. So that will help us focus some of our attention, such as through the Food Code as well as through some of us consumer messages about how to be more protective of public health.

So we will in fact be looking at ways to address those issues and we would certainly welcome comment on that as well. From the perspective of where we are from risk management, we now take this information under advisement.

As the comments come in on the risk

assessment, are reviewed and if they should in fact change some of the outcomes with regards to what was presented today then those two will be taken into account in the formulation of our policies.

DR. GOLDMAN: Okay. Thank you, Dr. Engeljohn. Anyone think of any last questions or comments before we completely close the meeting?

I do want to thank first of all, all of you for coming out today and spending the better part of a day with us listening to the scientific presentations of our risk assessments and for engaging us in the discussion for providing thoughtful comments and questions.

I want to thank all the presenters that are here - those here and those that are in the audience who presented this morning for making the day very informative for all of us.

Speaking of the questions and comments that we received today, in addition to the fact that the risk assessments themselves are posted on our Web site, the peer reviews and the responses to the peer reviews are also posted on our Web site.

At some point in the future the responses to your questions and comments will also be posted on the Web site. So this is an effort that the agency

has made and as it evolves into a more transparent process for producing and demonstrating it risk assessments and as you heard just a minute ago the comment period for these risk assessments is open until May 9.

I will point out that I went on the Web site this morning and looked at the docket and there have been 2,931 comments submitted to the docket with respect to the proposed rules. So there are a lot of comments for us to go through. Obviously we expect these risk assessments to provoke more comments and we, as Dr. Engeljohn said, will take those under advisement.

I also finally want to thank in addition the ladies this morning who were outside the room from our planning staff, Diane Jones, Sheila Johnson, and Mary Cutshall for making the arrangements for our meeting room and for greeting you as you come in.

I think that with that, unless there are any last comments or questions, we'll call the meeting adjourned. Thank you.

(Whereupon, the above-entitled matter was adjourned at 2:40 p.m.)